FILE NAME: P C PC

DATE:

DOC#: PC

DOCUMENT DESCRIPTION: L D D R A

IN THE CIRCUIT COURT

FOR ANNE ARUNDEL COUNTY

STATE OF MARYLAND,	:	
Plaintiff,	:	
-vs-	:	Civil Action No. 1109600
KEENE CORPORATION, et al.,	:	
Defendants.	:	

IN THE UNITED STATES DISTRICT COURT

FOR THE DISTRICT OF SOUTH CAROLINA

IN RE:	:	
Asbestosis Cases:	:	
Shipworker Insulator	:	
	:	

ACE REPORTING, INC.
216 E. 9th St. / Barrister House / 6th Floor
CINCINNATI, OHIO 45202
TELEPHONE: AREA CODE 513
241-3200

IN THE CIRCUIT COURT
STATE OF SOUTH CAROLINA
COUNTY OF RICHLAND

UNIVERSITY OF SOUTH CAROLINA,

Plaintiff,

-vs-

W.R. GRACE AND COMPANY, et al.,

Defendants.

Civil Action No. 85-CP-40-3789

CIRCUIT COURT

OF COOK COUNTY, ILLINOIS

Lavin v. Flintkote, et al.
Pauly v. Flintkote, et al.
Pyne v. Flintkote, et al.
Greaney v. Flintkote, et al.
Grant v. Flintkote, et al.
Healy v. Flintkote, et al.

IN THE UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF ILLINOIS
EASTERN DIVISION

- - -

CHARLES E. LYNCH and ELIZABETH LYNCH,	:	
Plaintiffs,	:	
-vs-	:	No. 85-C-8071
KEENE CORPORATION, et al.,	:	
Defendants.	:	

IN THE DISTRICT COURT OF THE STATE OF DELAWARE

IN AND FOR NEW CASTLE COUNTY

- - -

In Re: Asbestos Litigation	:	Master File No. Misc. 85-100
	:	Limited to:
	:	C.A. No. 85-03 (Morean) JJF
	:	C.A. No. 84-541 (Overkamp) MMS
	:	C.A. No. 85-61 (Brown) MMS
	:	C.A. No. 85-62 (Grady) MMS
	:	C.A. No. 82-79 (O'Neal) JJF
	:	C.A. No. 84-251 (Fogleman) JJF
	:	C.A. No. 85-550 (Williams) JJF

IN THE SUPERIOR COURT OF THE STATE OF DELAWARE

IN AND FOR NEW CASTLE COUNTY

In Re: Asbestos Litigation

C.A. No. 79C-DE-125 (Lee)
C.A. No. 81C-JA-79 (Blissick)
C.A. No. 81C-FE-27 (Mergenthaler)
C.A. No. 81C-OC-104 (Pannell)
C.A. No. 82C-MR-71 (George)
C.A. No. 82C-AP-77 (Conway)
C.A. No. 82C-AP-98 (Shewbrooks)
C.A. No. 82C-MY-30 (Murphy)
C.A. No. 82C-NO-70 (Muzzi)
C.A. No. 82C-JL-80 (Mancari)
C.A. No. 82C-DE-26 (Clark)
C.A. No. 82C-DE-27 (Sheppard)
C.A. No. 82C-DE-91 (Korecki)
C.A. No. 82C-MR-97 (McCarty)
C.A. No. 82C-DE-105 (DeHart)
C.A. No. 82C-DE-102 (Logan)
C.A. No. 82C-DE-140 (Stagg)
C.A. No. 83C-FE-108 (Millard)
C.A. No. 83C-MR-89 (Waseilewski)
C.A. No. 83C-AP-20 (Hunter)
C.A. No. 83C-AP-119 (Lloyd)
C.A. No. 84C-JA-86 (Cale)
C.A. No. 84C-FE-121 (Tull)
C.A. No. 84C-AP-72 (Huber)
C.A. No. 83C-MY-28 (McDaniel)
C.A. No. 84C-MY-132 (Davis)
C.A. No. 84C-MY-145 (Bradley)
C.A. No. 84C-JL-64 (Nichols)
C.A. No. 84C-JL-86 (Reynolds)
C.A. No. 84C-SE-34 (Moyer)
C.A. No. 85C-JA-42 (Hudson)
C.A. No. 85C-JA-52 (Kramarck)
C.A. No. 85C-JA-53 (Chasteen)
C.A. No. 85C-JA-121 (Dixon)
C.A. No. 85C-FE-10 (Farrall)
C.A. No. 85C-FE-25 (Bell)
C.A. No. 85C-FE-39 (Lipscomb)
C.A. No. 85C-AP-60 (Oliphant)
C.A. No. 85C-AP-115 (Woodlen)
C.A. No. 85C-MY-13 (Bjorson)
C.A. No. 85C-JN-29 (McGuire)
C.A. No. 85C-SE-119 (Oberle)
C.A. No. 85C-OC-12 (Summers)
C.A. No. 85C-OC-60 (Hanson)
C.A. No. 85C-NO-119 (Graham)
C.A. No. 85C-DE-135 (Jensen)
C.A. No. 85C-DE-120 (Hessler)
C.A. No. 85C-DE-145 (Piegalski)
C.A. No. 85C-DE-145 (Milewski)
C.A. No. 85C-DE-114 (Layer)
C.A. No. 86C-JA-14 (Dunn)

Deposition of ROBERT J. RITTERHOFF, M.D., a witness herein, taken by the plaintiffs as upon direct examination pursuant to all applicable rules of civil procedure and pursuant

to Notice to Take Deposition, at the Hamilton County Coroner's Office, 3159 Eden Avenue, Cincinnati, Ohio, at 9:00 a.m. on Thursday, January 30, 1986, before Betty J. Schwab, a notary public within and for the State of Ohio.

- - -

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I N D E X

WITNESS:	PAGE:
Robert J. Ritterhoff, M.D.	
Direct Examination by Ms. Cannon	4

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E X H I B I T S

PLAINTIFFS' RITTERHOFF EXHIBIT:	IDENTIFIED:
No. 1, a letter dated November 1, 1944	28
No. 2, a letter dated November 10, 1944	29
No. 3, a letter dated December 23, 1944	31
No. 4, a letter dated December 27, 1944	31
No. 5, a report dated November 20, 1944	31
No. 6, a copy of The American Journal of Pathology	31

ROBERT J. RITTERHOFF, M.D.

of lawful age, a witness herein, being first duly sworn as hereinafter certified, was examined and deposed as follows:

DIRECT EXAMINATION

BY MS. CANNON:

Q Dr. Ritterhoff, would you state your name and address?

MR. MONTGOMERY: Before we begin the deposition, can you get the representation of people on the record so we know who is doing what with whom or who we have here?

MS. CANNON: Assistant Attorney General for the State of Maryland.

MR. MONTGOMERY: And you're appearing in which case?

MS. CANNON: State of Maryland vs. Keene Corporation, et al. It's in the Circuit Court for Anne Arundel County, Maryland.

MR. MONTGOMERY: I just want names and cases, basically. That's good enough to start with.

MR. BRICKMAN: My name is Mike Brickman. I'm appearing here on behalf of the law firm of Blatt & Fales. We have plaintiffs in a number

of jurisdictions in which this has been noticed, including, but I'm not sure it's limited to, the United States District Court for the District of South Carolina, the United States District Court for the Northern District of Illinois, Eastern Division, and I'm not sure that's all, but I know it's at least in those cases, and, if there are others, I'll be sure that the court reporter gets those others.

MR. MONTGOMERY: Can you get us the names of those cases, the ones that you know of for now that have been noticed?

MR. BRICKMAN: In the District Court for the District of South Carolina, this deposition has been noticed for all the asbestosis cases. They have a caption there of In Re: Asbestosis Cases, and also it has been noticed in the State Court of South Carolina in The University of South Carolina case vs. W.R. Grace, et al., and in the Northern District of Illinois it has been specifically noticed in the case of Charles E. Lynch and Elizabeth Lynch vs. Keene Corporation, et al.

MR. MONTGOMERY: Thank you.

MR. MADEKSHO: My name is Larry Madeksho. I'm here on behalf of the plaintiffs in regard to various jurisdictions in the State of Texas.

MR. MONTGOMERY: Has there been a notice issued in those cases?

MR. MADEKSHO: No. I just became aware of the deposition, I think, Monday.

MR. MONTGOMERY: So there is no pending notice of deposition in this case?

MR. MADEKSHO: That is correct.

MR. BRICKMAN: Before we go on, I also have with me notices of depositions of Dr. Ritterhoff in the Superior Court of the State of Delaware in and for New Castle County, In Re: Asbestos Litigation, and in the District Court of the State of Delaware, In Re: Asbestos Litigation.

MR. MONTGOMERY: Would it be possible for counsel for the plaintiffs to give the court reporter copies of all of the notices in which this deposition is proceeding?

MR. BRICKMAN: That will be fine. I'm not sure these are all the cases in which it has been noticed. We will give her those to make a part of the record, if you like, and we will provide

her with copies of any other notices that were sent out.

MR. MONTGOMERY: My name is James J. Montgomery from the firm of Bloom & Greene in Cincinnati, Ohio. I'm here in property damage cases in which there is a legitimate notice issued for the Celotex Corporation and Carey Canada, with the exception of the Selah School District case in Washington, which I'm only here on behalf of Phillip Carey. In the personal injury cases in which there has been a legitimate notice issued, I'm here on behalf of the Wellington Group of defendants, except insofar as there are other counsel here that are specifically appearing for the Wellington Group in specifically noticed personal injury cases.

I would also like to specifically object to any use of this deposition in any case in which there is not a valid notice that has been timely filed to all parties in the case, in any case in which the Celotex Corporation or Carey is involved in the property damage aspects or the members of the Wellington Group are involved in a personal injury aspect.

Does that cover it?

MR. DEMICHELIS: I think so. On behalf of the Owens-Corning Fiberglas. I would speak to the same objections which Mr. Montgomery has made.

MR. MADEKSHO: Could you identify yourself, please?

MR. DEMICHELIS: My name is Clement J. DeMichelis.

MS. CANNON: Michaels?

MR. DEMICHELIS: DeMichelis. I join Mr. Montgomery in objecting to use of this deposition in any proceeding in which it has not been properly and timely noticed.

MR. ATKINS: I'm Jim Atkins with Robert H. Hood & Associates in Charleston, and I accept in part and parcel the objection raised by Mr. Montgomery as to notice. I'm here on behalf of the Wellington Group, as well as the property damage cases properly noticed for Keene Corporation.

MS. PORTER: My name is Ruth Porter. I'm here on behalf of W.R. Grace in South Carolina and Empire Ace. We would adopt Mr. Montgomery's

objection in its entirety.

MR. MARTIN: Ted Martin with Paxton & Seasongood in Cincinnati, here on behalf of Fiberboard in the property damage cases, and I join in Mr. Montgomery's objections.

MR. BURTCH: My name is John H. Burtch. I represent GAF Corporation, and I would join in Mr. Montgomery's objections.

MR. FRYE: Richard Frye from Columbus. I'm here in the Selah School District case for United States Gypsum pursuant to notice, and I join in the objection to the use of the deposition in any other case in which it's not noticed.

MR. STERN: Geoff Stern representing Combustion Engineering in the Selah School District. Same objection.

MR. LEONARD: Roy Leonard, Green & Green. We're representing Raymark Industries in the two cases that have been noticed, United States District Court for the District of South Carolina, In Re: Asbestosis Cases, State of South Carolina, etc., vs. W.R. Grace Company, and in the United States District Court, Northern District of

Illinois, Eastern District, Charles Lynch and Elizabeth Lynch vs. Keene Corporation, et al.

MR. HOLLINGSWORTH: I'm Michael Hollingsworth representing the Cincinnati Board of Education, here observing.

DIRECT EXAMINATION (CONTINUED)

BY MS. CANNON:

Q Dr. Ritterhoff, would you state your name and address?

A Robert John Ritterhoff, 123 Hosea, H-o-s-e-a, Cincinnati, 20.

Q Dr. Ritterhoff, is that your home address?

A Home address.

Q Do you have a business address?

A Not today.

Q Are you retired right now?

A Partially.

Q And you also do some work; is that right?

A I'm working five half days.

Q Where?

A At Deaconess Hospital, Straight Street, Straight and Clifton Avenue, Cincinnati.

Q Dr. Ritterhoff, where did you go to medical school?

A Here, Cincinnati, graduated '36.

Q After you graduated, did you do a residency?

A I interned, first.

Q Where?

A U.S. Marine Hospital, Stapelton, Staten Island, New York, then returned here to the department of pathology, University of Cincinnati. I guess that was '37, '38. I don't remember which. And there I remained until 1973.

Q Meaning that you remained in the department of pathology at the University of Cincinnati until 1973?

A Yes, in various capacities.

Q Did you do your internship in pathology?

A No. I did that -- it was a general internship.

Q Doctor, are you licensed to practice in Ohio?

A Right.

Q In any other state?

A I had reciprocity with Kentucky, but whether that is valid today, I don't know.

Q Doctor, are you Board certified?

A No.

Q Doctor, you said that, beginning in approximately 1937, you have been associated with the University of Cincinnati in various capacities?

A All right. I was sent from the department of pathology to the Hamilton County Tuberculosis Hospital on a part-

time basis, I guess in '38, and I was to continue my affiliation with the department of pathology. That continued until '47. In '47, I became childish, as I learned that the electrician at the hospital was making more than I was and quit Dunham, went to St. Elizabeth Hospital, Covington, Kentucky, full time, continued on a part-time basis in the department of pathology.

After 23 years at St. Elizabeth, I was fired, and then I returned, I guess in '70, on a temporary appointment to the department of pathology while they were looking for a new departmental head, remained there until '73. In '73, the new departmental head decided he didn't need my services. I then went to the Hamilton County Coroner's Office and remained there until '79, being retired. A month later, I began work at Deaconess Hospital, part time.

Q Doctor, I would like to back up and ask you a couple of questions. You said that, when you began at the Hamilton County Tuberculosis Hospital, that you continued on a part-time basis in the department of pathology at the University of Cincinnati?

A Right.

Q And what were you doing at the University of Cincinnati?

A Teaching.

Q What were you teaching?

A. Pathology.

Q. Were you full time at Hamilton County Tuberculosis Hospital?

A. Eventually, I became full time, with the understanding that I was to continue at the medical school.

Q. Do you remember approximately what time period you became full time at Hamilton?

A. No, gosh, no.

Q. And what were your duties at Hamilton County Hospital?

A. I ran the laboratory.

Q. Were you the head of the laboratory?

A. Yes, ma'am.

Q. Who was your immediate superior?

A. Originally, it was Dr. Dunham, and then followed by Dr. Skavlem, John.

Q. What year was it that Dr. Skavlem became your immediate supervisor?

A. I don't remember.

Q. Do you know what his title was when he was your immediate supervisor?

A. Well, he would have been the medical director. The medical director of the Hamilton County Tuberculosis Hospital was appointed by the University of Cincinnati.

Q. When you ran the laboratory at the Hamilton County Tuberculosis Hospital, did you also supervise students from the University of Cincinnati Medical School?

A. They came out there, yes, um-hum.

Q. And worked in your laboratory?

A. No. They came out for demonstrations and lectures.

Q. Lectures by you?

A. By the various members of the staff.

Q. Exactly what were your duties in running the laboratory?

A. Well, one would have been the autopsy service. They were running around 100 autopsies a year. Usually, I had the help of a resident on that. And, then, the other problems were teaching medical students and then supervising the departments of chemistry and microbiology, hematology.

Q. You were supervising those departments at the hospital?

A. Yes.

Q. You said that part of your duties included conducting about 100 autopsies a year?

A. Right.

Q. How did you determine which bodies you were going to autopsy?

A. That was determined for me by the doctor who

requested the autopsy.

Q. Were there any -- what doctors requested the autopsies for you? Would this have been across the whole city of Cincinnati?

A. No, just the patients at Dunham Hospital or the Hamilton County Tuberculosis Hospital.

Q. Doctor, Dunham Hospital is a different hospital from the Hamilton County Tuberculosis Hospital?

A. No. The name was changed to Dunham. I don't remember the year.

Q. Did you autopsy all patients who died in the Dunham Hospital?

A. No. You autopsied only those patients on whom you had a permit.

Q. When you left Dunham Hospital in 1947 to go to St. Elizabeth Hospital --

A. Yes?

Q. -- what was your title at St. Elizabeth?

A. Director of the laboratory.

Q. Is St. Elizabeth Hospital still around?

A. Yes.

Q. When you went there in 1947, was it a specialized hospital?

A. It was a general hospital.

Q Did it remain a general hospital up until the time you left in 1970?

A Right.

Q Was that when you left?

A Yes.

Q And what were your duties at St. Elizabeth Hospital?

A The same as I gave you for Dunham Hospital, run the goddamn laboratory.

Q So that your duties, once again, involved doing autopsies?

A Autopsies.

Q Approximately how many autopsies did you do a year at St. Elizabeth?

A By '55, I think we were up to around 240 a year, for which I had resident help, and then it declined progressively. I don't know what the number was the last year I was there, which has been the story of most hospitals.

Q The number of autopsies has been declining?

A Yes.

Q In 1970, when you became full time at the department of pathology at the University of Cincinnati -- is that right?

A Yes, on a temporary appointment.

Q. Before we get to that, from 1947 to 1970, while you were full time at St. Elizabeth Hospital, you continued in your part-time capacity at the University of Cincinnati?

A. Right.

Q. And that would have been teaching?

A. Teaching.

Q. In 1970, were you temporary department head?

A. No.

Q. What was your position?

A. Associate professor.

Q. And did you have any duties other than teaching?

A. No.

Q. What was your title from beginning, when you joined the Hamilton County Coroner's Office in '73?

A. Then I became known as a deputy coroner.

Q. Who was the coroner in 1973?

A. Dr. Frank Cleveland.

Q. Did you remain deputy coroner until you retired in '79?

A. Right.

Q. Do you have a title right now in your part-time position at Deaconess Hospital?

A. No.

Q. You began there in 1979, in your part-time

position?

A. Right.

Q. And what are your duties there, or what were your duties there when you began part time?

A. My duties there are primarily to do with the description of material removed from patients at surgery. I take the specimen block, describe it, get the pertinent material out of the specimen to start the processing of microslides.

Q. Doctor, you know we can take a break at any time when you want to. Just let me know.

Have you published any articles?

A. Yes, but I don't have a list of them with me. I have, yes.

Q. Approximately how many?

A. Oh, maybe 15.

Q. Are you currently a member of any professional organizations?

A. I'm a retired member of the Academy of Medicine of Cincinnati, the State of Ohio, the AMA, the American Society of Clinical Pathologists, the American Association of Pathologists and Bacteriologists, and a retired member of the Academy of Forensic Scientists.

Q. Were you an active member of each of those organizations before you retired?

A. Right.

Q. Were there any other organizations that you were an active member of that you are not presently a retired member of?

A. Like what?

Q. Well, any other organizations, other --

A. You mean professional?

Q. Yes, professional organizations other than those you have already named.

A. The only other one is the honorary medical, Alpha Omega Alpha.

Q. Doctor, are you familiar with asbestosis?

A. I became familiar with asbestosis with this case of Mr. Scobie in 1944. Prior to this time, my knowledge was, at most, fragmentary. With studying the autopsy material on Mr. Scobie, I had to resort to the literature.

Q. Doctor, before we get to Mr. Scobie, I want to ask you: When was the first time that you heard of asbestosis?

A. Oh, hell, I don't know. That could have been in medical school. It could have been after medical school. I don't know.

Q. Was it, well, before 1944, when you said that you had Mr. Scobie's case?

A. I would say that I was dimly aware of it. I hadn't

seen it.

Q You had read about it, though, before then; is that right?

MR. FRYE: Objection.

A Not particularly.

Q Doctor --

A You didn't let me finish.

Q Sorry. My question was: Had you heard about it before --

A And I'm telling you that my knowledge came with Mr. Scobie.

Q I'm going to ask you about Mr. Scobie, Doctor.

MR. MONTGOMERY: I'm sorry. This witness has an absolute right to finish any answer he thinks --

MS. CANNON: I think he is finished. Thank you.

MR. MONTGOMERY: Doctor, if you want to finish, you have that right.

A No, I'm not finished.

Q Doctor -- okay.

A Just a minute.

Q Excuse me. Wait. I don't think there's a question pending. My question was whether or not you had had

any knowledge before 1944, and I believe your answer was that your knowledge before 1944 was fragmentary, and, at that point in time, you were fixing to tell me about Mr. Scobie. I have not asked you about Mr. Scobie.

MR. MONTGOMERY: I'm sorry, Counselor. The witness has indicated that he is not finished with his answer, which you interrupted him earlier, and I believe we owe him the courtesy of allowing him to finish any answer that he feels he has incompletely answered.

MR. DEMICHELIS: Counselor, this is your witness. I don't think you have the right to cross-examine this witness or direct the witness.

MS. CANNON: I'm not cross-examining the witness.

MR. DEMICHELIS: You're sure making a pretty good effort at it right now.

MS. CANNON: There is no question pending.

MR. BRICKMAN: Let's go off the record.

MR. DEMICHELIS: If you want to give an explanation to the doctor, let's do it on the record.

MR. BRICKMAN: Let me explain to you, there

are certain procedures that go on in court, where you have to ask a certain question and it has to be answered in a certain way, and you have to lay a predicate or a foundation. Sometimes, it's very technical, but if you answer, giving additional information other than what was asked, some of the attorneys may move to strike that testimony, and we would have to reask it. That is why she needs just to get, sometimes, an answer to the question, and she'll follow it up. Or, if you want to explain it further, you, of course, have that right, but, sometimes, she needs just the yes or no, and then the explanation with it, just for procedural point.

MR. DEMICHELIS: I'm wondering who appointed you judge.

THE WITNESS: Would you read back the question?

MR. BRICKMAN: Did you have an objection?

MR. DEMICHELIS: I object to your instructing this witness on anything, sir. He's a witness being questioned on direct examination. Plaintiffs' counsel in this case had a fair amount of time, before this deposition started, to confer

with the witness. None of the defense counsel did. I hadn't seen him before I walked into this room. Now, if you haven't got him prepared to give whatever testimony he's going to give on direct, we'll just have to go with what we have got.

MS. CANNON: We'll clarify one thing, that, perhaps, Mr. -- the person speaking, I don't know. You say your name is Mr. Montgomery?

MR. DEMICHELIS: No. My name is DeMichelis. That's a little more difficult.

MS. CANNON: Mr. DeMichelis has not spoken to the doctor before we began, but there are other defense counsel that have spoken to him.

MR. DEMICHELIS: I'm not worried about them.

MS. CANNON: I want that on the record. After we spoke with the doctor, we left the room. Defense counsel conferred, and we did not come back in until we were told that the defense counsel had finished their conferring with the doctor.

THE WITNESS: Well, let's get on with the damn thing.

MR. MONTGOMERY: Doctor, I think you have

the right to finish an explanation if you feel --

MS. CANNON: There is no pending question.

MR. DEMICHELIS: Doctor, you don't have to do anything unless the judge tells you.

MR. MONTGOMERY: He has the right, and, Counsel, I think you're being unfair in trying to suggest to him he doesn't have the right.

MS. CANNON: Mr. Montgomery --

MR. MONTGOMERY: Please, can I finish? If he is not comfortable with an answer that he has given and feels that it requires additional explanation, this witness has that absolute right in every jurisdiction in this country, and I don't think your suggestion to him to the contrary is anything other than unfair.

MS. CANNON: Okay. Mr. Montgomery, you'll have an opportunity to ask him questions. Let's go ahead and go on to the next area.

DIRECT EXAMINATION (CONTINUED)

BY MS. CANNON:

Q. Doctor, before 1944, had you heard of asbestos in connection with cancer?

A. I am not sure that I had heard of that.

MR. DEMICHELIS: I want to make a point for the record here.

MR. BRICKMAN: Is this an objection?

MR. DEMICHELIS: It's not an objection, because I don't know what the situation is in all these cases. I want to make the point that, if this last question suggests that this witness might be being called by plaintiffs as an expert witness and that the plaintiffs intend to use this deposition on trial to give expert testimony, to the extent that, in any of these cases, this witness has not been previously noticed or identified as an expert witness, I'm going to make an objection to any testimony in that area.

MR. MADEKSHO: I think those objections are in the record already.

MR. DEMICHELIS: I'm not aware. That happened today, this morning, sir?

MR. MADEKSHO: That is correct.

MR. DEMICHELIS: I haven't seen that objection this morning, and I thought I was here since things started. The specific point I'm making is not with respect to notices that we discussed earlier, but with respect to the

utilization of this witness as an expert witness to the extent that -- not with respect to whether we received notice of the taking of his deposition, but with respect to whether he had previously been identified as an expert witness and defendants given an opportunity to discover his testimony by way of an earlier deposition before this meeting to get his testimony at a testimonial deposition to be read in court. I hope that is clear, gentlemen.

DIRECT EXAMINATION (CONTINUED)

BY MS. CANNON:

Q Dr. Ritterhoff, did you perform an autopsy on a Herbert C. Scobie?

A I did.

Q This was in about 1944 that you performed the autopsy?

A Yes.

Q Do you remember who the referring physician was?

A Dr. John Skavlem.

Q And this would have been while you were at Hamilton Tuberculosis Hospital?

A Right.

Q Did Dr. Skavlem give you any information when he

referred the Scobie case to you?

A Only that it was his -- as I remember, his only case of asbestosis, and that he had died, and that he was interested in seeing the findings.

Q After you conducted the autopsy, did you send some of your findings to Dr. Gardner?

A I did.

Q And referring to Dr. Leroy Gardner, how did you come to know him?

A I previously had had correspondence with Dr. Gardner in regard to a case of acute silicosis that had developed in an employee of Procter & Gamble, and I knew that Dr. Gardner had described acute silicosis in the Gaulley Bridge tunnel workers. And so, on the basis of that knowledge, I sent slides of the case to Gardner.

Q Now, referring slides of the case to Gardner, are you referring to --

A To the acute silicosis case.

Q This was before 1944?

A Yes.

Q Okay. Now, and then, in 1944, with Mr. Scobie, you also sent some information to Dr. Gardner; is that right?

A Right.

MR. MONTGOMERY: I object to leading

the witness.

Q. Did you have any contact with Dr. Gardner in between the time that you sent him the information on the silicosis case and the information on Mr. Scobie?

A. There was a personal contact, and Dr. Gardner visited Dunham Hospital or the Hamilton County Tuberculosis Hospital, and I think Dr. Dunham, who was then the medical director, brought Gardner to town, and he spent the afternoon with us, the staff, at the TB hospital talking about various aspects of lung disease, and it may be, at that time -- I have forgotten -- that I had told him that I was unfamiliar with the asbestos body, and he subsequently sent me a couple slides showing me the asbestos body.

Q. Dr. Ritterhoff, when you said you were unfamiliar with the asbestos body, that means you hadn't seen it on slides?

A. I hadn't seen it, no.

MS. CANNON: Could we have this marked, please, as Ritterhoff Exhibit No. 1?

(Plaintiffs' Ritterhoff Exhibit No. 1, a letter dated November 1, 1944, was marked for identification.)

Q. Dr. Ritterhoff --

MR. MONTGOMERY: Excuse me. Could you give us just a second to look at this?

(Off the record.)

MR. BRICKMAN: Have you all had an adequate opportunity to examine it?

MR. MADEKSHO: It would be a good idea to have the entire series of exhibits marked and ask the doctor if he is familiar with them.

(Plaintiffs' Ritterhoff Exhibit No. 2, a letter dated November 10, 1944, was marked for identification.)

MR. MADEKSHO: Doctor, we want to make sure -- there's a series of four documents that we want you to have the opportunity to look at this morning instead of, perhaps, passing them on down the table to other counsel. If they need some additional time to review these other copies, we will give them all the time that they need, if they so desire.

MR. MONTGOMERY: Pardon me, Mr. Madeksho. Since you were not here --

MS. CANNON: Excuse me for just a second. I'm sorry. Could we go off the record?

(Off-the-record discussion.)

MR. MONTGOMERY: I was in the process of making an objection. Mr. Madeksho, I hope I

have your name right.

MR. MADEKSHO: Madeksho (pronunciation).

MR. MONTGOMERY: Please don't take this personally, but you have indicated that you are not here in any case in which there is a notice pending, so I don't think it's proper for you to make any objections or speak on the record, since there is no case you are noticed in.

MR. MADEKSHO: I'm here representing other plaintiffs in the cases in Texas and the Eastern District and the Southern District of Texas, and we can make objections on the record as we see fit.

MR. MONTGOMERY: But you did concede and tell us, at the beginning, that you are aware of no notices in which any of those cases are here.

MR. MADEKSHO: I'm also here as co-counsel in regard to --

MR. BRICKMAN: Certain cases in South Carolina, he is also supposed to be here.

MR. MONTGOMERY: That sounds to me like an ad-lib, right after you whispered in his ear, and, Counsel, I think that's a little bit of a

boot strap. Let's be realistic about it.

MR. BRICKMAN: That is not correct, and, if this was a boot strap, that wouldn't make a difference.

MS. CANNON: I want to go off the record for a minute so I can finish marking these documents.

(Plaintiffs' Ritterhoff Exhibit No. 3, a letter dated December 23, 1944; No. 4, a letter dated December 27, 1944; No. 5, a report dated November 20, 1944; and No. 6, a copy of The American Journal of Pathology, were marked for identification.)

DIRECT EXAMINATION (CONTINUED)

BY MS. CANNON:

Q Dr. Ritterhoff, I'm giving you a copy of what has been marked as Ritterhoff Exhibit No. 1. Have you ever seen that before?

A I must have, but I don't remember this.

Q Down at the bottom, there is a signature. Do you recognize that signature?

A That's mine.

Q That's your signature?

A Yes.

Q Okay. And what is Ritterhoff Exhibit No. 1?

MR. MONTGOMERY: Note my objection. He said he doesn't recognize it. Anything else beyond that, it would be inadmissible. I think it would speak for itself, as to what it is.

Q Dr. Ritterhoff --

A It speaks for itself, yes. I must have written it.

Q Okay. I understand that. Now, what is it?

A Well, it's a letter.

MR. MONTGOMERY: Same objection.

A A letter to Dr. Gardner.

Q Um-hum.

A And I'm asking for help.

Q What's the date on the letter?

A November 1, 1944.

Q Let me show you Ritterhoff Exhibit No. 2.

A This is a response from Dr. Leroy Gardner to me, received or written November 10, 1944.

Q Okay. And Ritterhoff Exhibit No. 3, on the second page of Ritterhoff Exhibit No. 3, there's a signature. Do you recognize that signature?

A That's my signature.

Q And what is Ritterhoff No. 3?

A Well, it's a letter that I wrote to Dr. Gardner.

Q And what's the date on the letter?

A December 23, 1944.

Q Thank you. I ask you to look at Ritterhoff Exhibit No. 4. What is that?

A It's a letter from Dr. Leroy Gardner, sent December 27, 1944.

Q To who?

A To me.

Q I show you Ritterhoff Exhibit No. 5. Have you ever seen Ritterhoff Exhibit No. 5 before?

A Yes. I think that was in material you sent to me originally.

Q Did you ever see it before I sent it to you?

A I don't know. I don't know that.

Q Doctor, let me show you Ritterhoff Exhibit No. 6. What is that?

A Well, this is a reprint of the article I wrote concerning the findings in Mr. Scobie, The American Journal of Pathology.

Q Now, Doctor, going back to Ritterhoff Exhibit No. 5, I'm going to ask you to turn to -- and, you have, actually, the original, the reprint, I guess, that you got at the time the article was published, of Exhibit No. 6; is that

right?

A Yes, yes.

Q Would you turn to page 496 of that article?

A Right.

Q Down at the bottom is a paragraph that begins "More frequently."

A Yes.

Q There's a part in that paragraph that reads, "Dr. Leroy U. Gardner, who also studied this case, stated that these bodies stained," and then there's a quote. Would you read the quote?

A "These bodies stained 'red with acid fuchsin of van Gieson-Weigert instead of black like elastic tissue' and that 'von Kossa's calcium stain is negative.'"

Q Thank you very much. Now, Doctor, I'm going to ask you, also now, to look back again at Exhibit 5.

A Exhibit 5, all right. Now, where are we?

Q Okay. Exhibit 5 is dated November 20, 1944. It says "received," and it has Herbert Scobie on top.

A Yes.

Q Now, would you read -- I'm in the third full paragraph, the second sentence. Would you read that sentence?

MR. MONTGOMERY: Note my objection to his reading from the document, which has not been

identified. I think that's a highly irregular procedure by the rules of evidence in practically every jurisdiction I'm aware of.

MR. MADEKSHO: I believe we are in the process of getting it identified.

Q Doctor, would you read that, please?

MR. DEMICHELIS: You mean into the record or for himself?

MS. CANNON: No. I want him to read it out loud.

MR. DEMICHELIS: Then, I object.

A "These stain black with hematoxylin, red with the acid fuchsin of van Gieson-Weigert instead of black like elastic tissue. Von Kossa's calcium stain is negative."

Q Thank you, Doctor. Do you know where you got the quote from in your article, which has been marked as Exhibit 6, that you just read?

MR. MONTGOMERY: Note my objection.

MR. DEMICHELIS: I also object, because I think this procedure constitutes cross-examination.

MR. ATKINS: For the record, instead of each defense counsel making an objection, at this point, any defense counsels' objections will be anticipated, unless there is some other

reason, with all the other defense counsel.

MR. DEMICHELIS: I want to add a reason.

MR. BRICKMAN: You do not agree with that, what Mr. Atkins said?

MR. DEMICHELIS: Certainly, I think I said I did. Didn't I?

MR. BRICKMAN: I'm sorry. I just misunderstood.

MR. DEMICHELIS: You're batting 500. You were right once and wrong once.

A Well, obviously, if I may speak --

Q Yes, I want to know where you got the quote from, yes.

A I must have had this, because there are cross-references here.

Q Okay. When you say you must have had this, you're saying you must have had Ritterhoff Exhibit No. 5?

A Yes. I must have had it sometime.

Q Sometime before you wrote the article which is Exhibit 6?

A Possibly, um-hum.

MR. MONTGOMERY: Move to strike all reference to Exhibit 5.

MS. CANNON: Denied.

MR. MONTGOMERY: I appreciate your prompt ruling.

Q Dr. Ritterhoff, did you ever speak to anyone at Phillip Carey concerning the Herbert Scobie case?

A I spoke with one man.

Q When was that in relationship to when your article was written, Exhibit 6, if you know?

A I would say that it was after everything was written up.

Q After everything was written up, meaning after what things were written up?

A The paper, whether it was after publication or before publication, I don't know.

Q Where did you speak with this -- you said you spoke with someone from Phillip Carey. How do you know it was a Phillip Carey person?

A I went to the Phillip Carey office, and the office was out at the plant.

Q Where was the plant located?

A Well, it's out in -- what is it -- Loveland or Lockland or whatever it is.

Q In Cincinnati?

A Yes.

Q Why did you go there?

A It was suggested to me by Dr. Skavlem.

Q Okay. Who did you speak with at the Phillip Carey plant?

A I don't know the man. I don't remember the name. I'm sure I was told the name, but I don't remember it.

Q Where in the plant did you speak to the person?

A In his office.

Q Was there anyone else in the office?

A No.

Q And how long did the conversation last?

A Fifteen, twenty minutes.

Q And what did you tell him?

A I told him that Mr. Scobie died of asbestosis.

Q What did this person look like?

A He was a kindly gentleman, gray-haired. That's all I remember.

MS. CANNON: I would like to take a minute off the record.

(A recess was taken.)

DIRECT EXAMINATION (CONTINUED)

BY MS. CANNON:

Q Dr. Ritterhoff, where did Mr. Scobie work?

A Where did Mr. Scobie work?

Q Um-hum.

MR. MONTGOMERY: Objection. That's for the record only.

A Well, according to information that I received, he worked at Phillip Carey.

Q At what plant or where at Phillip Carey?

MR. MONTGOMERY: Same objection. Hearsay.

A That, I don't know. All I know is he was an employee of Phillip Carey.

Q Were you given any information concerning a compensation claim filed by Mr. Scobie's widow?

A No.

MR. MONTGOMERY: Objection.

Q Did you discuss any compensation from Phillip Carey to Mr. Scobie's widow with anyone?

MR. MONTGOMERY: Objection.

A Do I answer or not?

Q Yes, sir.

MR. BRICKMAN: Yes, sir, you go ahead and answer.

A The only information I received was from Dr. Skavlem, and he told me that the widow had been privately and adequately compensated.

MR. MONTGOMERY: Move to strike on the basis of hearsay evidence and not responsive.

Q Dr. Ritterhoff, the article that you wrote that we referred to earlier as Exhibit 6 --

A Yes?

Q -- this article discussed the Scobie case; is that correct?

A Right.

Q And did you do research in preparation for writing this article?

A What do you mean, research?

Q Did you go to the library and get any articles to read?

A Yes.

Q And I see you're turning -- you're opening up the article right now?

A Yes. And these would be some of the things that I read. Some 29 articles were quoted from.

Q And when you say these, you're referring to what's the list of references on page 504 and 505 of the article?

A Right.

Q Now, where did you get these articles?

A Well, I suppose -- well, I did spend time in the medical library.

Q Would you look at the third article?

A Yes.

Q What's the name of that article? I'm sorry -- the name of the magazine?

A It's Deutsche med. Wchnschr volkenschrift.

Q Where did you locate that article or that magazine?

A It was cited. I have it cited by Egbert, and Egbert is number five, pulmonary asbestosis. Report of a case with necropsy findings, American Review Tuberculosis.

Q Did you have any access, yourself, to any foreign journals?

A Certainly, the British ones are over at the medical school library.

Q Would you look at reference number one?

A Yes?

Q Where did you locate that article?

A Gloyne's article was in the American Journal of Public Health, so that's a national journal here.

Q If you needed to get a foreign medical journal, what would you do?

A I would merely tell the librarian, and she would get it, if they didn't have it.

Q Were there some that were already in the library?

A I don't remember asking her to do that. Most of these were -- well, these are available in the library.

Q So, here, on four, there's a reference to a

British medical journal?

A Yes. That's over there. That's over there, yes.

Q And you say that's over there. It would have been over there, meaning in the medical library?

A Medical school library.

Q At the time the article was written?

A Right.

Q For publication in 1946?

A Right, yes.

Q I'm almost done, Doctor. Dr. Ritterhoff, let's go back to Exhibits 1 and 3. Exhibits 1 and 3 are those letters that you sent to Dr. Gardner concerning Scobie?

A Right.

Q And 2 and 4 are letters that you received from him in reply to those?

A Yes.

Q Now, did you rely upon the information in these letters in writing your article, Exhibit 6?

A In part, I'm sure. I don't know --

Q Doctor, I don't think there is any question pending.

MR. MONTGOMERY: If you want to explain a prior answer, Doctor, please do. You have the right to explain anything you feel is --

MS. CANNON: Just a minute. I think we can go back and read what the question is.

THE WITNESS: No. You're just going to have to keep quiet.

MS. CANNON: Excuse me, Doctor.

THE WITNESS: You're going to have to excuse me.

MS. CANNON: Doctor, there is no question pending.

MR. DEMICHELIS: The doctor apparently wants to complete his answer.

MS. CANNON: Mr. Montgomery is going to have an opportunity to be able to ask you some additional questions.

MR. DEMICHELIS: Wait a minute. If you're going to instruct the witness not to say something, we're going to have to ask that this deposition be recessed until we get a ruling from a court on this. I don't think any counsel has the right to tell a witness to shut up at any time. I know of no rule in any jurisdiction that permits that procedure.

MS. CANNON: Actually, there is a rule in Maryland that permits that procedure.

MR. DEMICHELIS: Whether or not his explanation is necessary, the court will later decide whether the court wants to strike it, and you'll just have to listen to it. Doctor, you say what you want to say.

THE WITNESS: All right. I merely want to say that I was very fortunate in having available a man such as Dr. Leroy Gardner, who served as a consultant for me, who really went out of his way. I don't know where I would find a man like that today. That's all I wanted to say.

MR. MADEKSHO: Thank you, Doctor.

MS. CANNON: Thank you, Doctor. That's very helpful. Nothing further.

MR. BRICKMAN: Excuse me. Hold on a second.

DIRECT EXAMINATION (CONTINUED)

BY MS. CANNON:

Q Now, when you went to the Phillip Carey plant, Doctor, you said you went sometime around the time that your article was being published?

A No. I don't know the exact time. I would say it was when everything was worked up, written up. Whether or not it was before or after this publication, I don't know.

Q Was it within six months of the publication,

either one side or the other?

A I don't know.

Q Was it within a year of the publication?

A I don't know.

Q Do you know how close to the autopsy of Mr. Scobie it was?

A I would say it was fairly close.

Q And fairly close, meaning within six months of the autopsy?

A I would guess.

Q Now, you said, when you went to the Phillip Carey plant and you spoke with this individual, whose name you don't remember, you spoke about Mr. Scobie?

A Right.

Q And that was the person -- you told the Phillip Carey person that Mr. Scobie had asbestosis?

A Right.

MR. MONTGOMERY: Note my objection to the leading nature of the question. Move to strike the response.

Q Okay. When you went to the plant and talked to the Phillip Carey person, what did you talk about?

MR. MONTGOMERY: Objection. Asked and answered.

MR. MADEKSHO: You can answer the question, Doctor.

A The purpose of the visit was to tell someone at Phillip Carey that their employee, Mr. Scobie, died of asbestosis.

MS. CANNON: Thank you very much. I have no further questions.

MR. BRICKMAN: I have no questions at this time.

MR. MONTGOMERY: Could we have a few moments? Could we break for a second?

(A recess was taken.)

MR. MONTGOMERY: Dr. Ritterhoff, I thank you for coming. I have no questions.

(Other defense counsel stated that they had no questions.)

ROBERT J. RITTERHOFF, M.D.

- - -

DEPOSITION CONCLUDED AT 10:30 A.M.

C E R T I F I C A T E

STATE OF OHIO :
: SS
COUNTY OF HAMILTON :

I, Betty J. Schwab, the undersigned, a duly qualified and commissioned notary public within and for the State of Ohio, do hereby certify that before the giving of his aforesaid deposition, the said ROBERT J. RITTERHOFF, M.D., was by me first duly sworn to depose the truth, the whole truth and nothing but the truth; that the foregoing is the deposition given at said time and place by the said ROBERT J. RITTERHOFF, M.D.; that said deposition was taken in all respects pursuant to Notice to Take Deposition; that said deposition was taken by me in stenotype and transcribed into typewriting under my supervision; that the transcribed deposition was submitted to the witness for his examination and signature; that I am neither a relative of nor attorney for any of the parties to this cause, nor relative of nor employee of any of their counsel and have no interest whatever in the result of the action.

IN WITNESS WHEREOF, I hereunto set my hand and official seal of office at Cincinnati, Ohio, this ____ day of _____, 1986.

MY COMMISSION EXPIRES:
January 29, 1991

Betty J. Schwab
Notary Public - State of Ohio

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PLAINTIFF'S EXHIBIT #6 Ritterhoff

COEXISTENT PULMONARY ASBESTOSIS AND SARCOIDOSIS*

JOHN H. SHAVLEIK, M.D., and ROBERT J. RITTMANN, M.D.
(From the Percy Shields Laboratory† of Deaconess Hospital, Cincinnati 5, Ohio)

Pulmonary asbestosis, regarded as a "modern disease" by Cloyne and Morewether,[1] was first described by Murray[2] in 1900. Although Fabr[3] described a case in 1914, interest in this disease was not reawakened until the case of Cooke and McDonald was described in 1927.[4] Since that time, there have appeared in the available literature reports upon approximately 150 necropsies on cases of pulmonary asbestosis.[5-12] The paucity of proved cases, in comparison with those of silicosis, is not due wholly to failure to report such cases, for in large necropsy series asbestosis is apparently of infrequent occurrence.[6-13] Further, despite the widespread usage of asbestos products, there are comparatively few people engaged in the asbestos industry. As of October, 1944, only 19,700 people were employed in this industry in the United States.[14]

Much has been written about the clinical, roentgenologic, and biopsic aspects of sarcoidosis. However, because of the infrequency and relatively benign character of this disease there are only isolated detailed necropsy reports. From the available literature there have been found only 53 reports of necropsies on cases of sarcoidosis.[15] Most of these were summarized by Pinner.[16]

These two diseases present many clinical and roentgenographic similarities, and, also, their more frequent fatal complications are alike: pulmonary tuberculosis and cardiopulmonary insufficiency. Bronchogenic carcinoma, a frequent complication of pulmonary asbestosis, has not, however, been described as associated with sarcoidosis. Likewise, there has not been a previous description of asbestosis with coexistent sarcoidosis. It is the purpose of this report to present the findings in such a unique case, the only example of either pulmonary asbestosis or sarcoidosis in a series of 1870 necropsies done at this hospital.

REPORT OF CASE

The patient was a white male, 42 years of age. Subsequent to hemorrhoidectomy in December, 1943, he had noticed that slight activity produced shortness of breath. He did not experience nocturnal dyspnea and he was able to lie flat in bed with no respiratory difficulty. There was no history of cough, hemoptysis, or chest embarrassment. Notwithstanding a good appetite and the absence of systolic

* Received for publication, May 9, 1945.
† Established and maintained by a grant from the Charles Phelps Taft Memorial Fund.

symptoms, there was a weight loss of 22 pounds from December, 1943, to March, 1944, at which time he presented himself for medical care.

The patient had worked in an asbestos plant for 25 years, the last 10 years having been in a supervisory position. During this entire time he had worked in one department in which asbestos pipe was made. There was a slight but appreciable dust hazard associated with the sawing and splitting of the dried asbestos pipe, despite precautionary exhaust ventilation. The total time the patient had spent upon this final operation of sawing was unknown; nor was it learned whether he had been negligent in using the provided respirators. To the company's knowledge, this was their first case of asbestosis.

Physical examination revealed the following findings: Temperature, 37° C.; respiration, 22 per minute; arterial blood pressure, 105/70 mm. Hg; height, 170 cm.; weight, 67 kg. The chest was of increased anteroposterior diameter. Respiratory excursions were equal but decreased. The percussion note was resonant and auscultation revealed fine rales over the bases of the lungs, posteriorly. There were no evidences of cardiac enlargement, irregularity, or decompensation. Cyanosis and clubbing of the fingers were absent. The liver was barely palpable.

Report on the roentgenogram of the chest (Fig. 1) was as follows: "The bony framework is normal. The trachea is in the midline. The hilum shadows are moderately enlarged, bilaterally. One small calcified area is present in each hilum. There are numerous small nodular densities scattered throughout both lung fields, especially throughout the lower lobes. There is some confluence of these densities in the left lower lobe. Emphysema is present."

Examination of the blood showed erythrocytes, 5.5 million; leukocytes, 6.6 thousand; 73 per cent neutrocytes; 21 per cent lymphocytes; 5 per cent monocytes; 1 per cent eosinophils; sedimentation rate, 26 mm. No abnormality was found in the urine. The vital capacity, 2100 cc., was 51 per cent of normal. Tuberculin tests were not done.

The patient was seen at regular intervals and his only complaint was increasingly severe exertional dyspnea. A roentgenogram of the chest 4 months after the initial chest film revealed no new findings. Although the patient greatly limited his activities, dyspnea became progressively more severe so that eventually, even at bed rest, there was extreme air hunger. At no time were there evidences of cardiac failure. He died approximately 11 months after the onset of symptoms, apparently from respiratory failure.

Autopsy Findings

The necropsy was performed 5 hours after death. Superficially, there was considerable decrease in the subcutaneous tissues and the body musculature. There was no clubbing of the nailbeds or dependent edema. The mediastinum was in the midline. Each lung completely filled its hemithorax and extended far into the anterior mediastinal space. The domes of the diaphragm, anteriorly, were at the level of the fourth interspace and fifth rib, right and left respectively.

The lungs were encased in markedly thickened, tough, yellowish white, generally fused pleurae. The interlobar fissures were obliterated by easily broken adhesions. Lobation was normal. Hemorrhagic fibrinous material, present over the posterolateral aspect of the left lower lobe, loosely bound the thickened parietal pleura to the lung in this area.

The frontal section of the left lung (Fig. 3) revealed coarse, lacy, tannish brown, hypercrepitant tissue throughout both lobes. Innumerable slightly elevated, grayish green, irregular, firm nodules, 1 to 2 mm. in diameter, were present throughout the lung. Thin, radiating, fibrous bands surrounded and connected these nodules. Also, slightly thickened pleural septa extended into the lung substance for variable depths. In the lung tissue about the bronchi of the second and third interspaces these nodules were somewhat confluent and a similar change was noted in the subpleural tissues for a depth of 3 to 5 mm. Generally, these nodules, present in moderate numbers, were separated by wide zones of dry emphysematous lung tissue studded by numerous minute, grayish tubercles. Dissection of the bronchi of the lower lobe disclosed that they were moderately dilated, cylindrically and saccularly, and lined by glistening white mucosa.

In the right lung the same changes were observed as were present throughout the left. However, the grayish green, irregular nodules tended to be more numerous, larger, and more confluent. This was particularly true of the anterior portions of the lower and middle lobes. Also, the nodules were connected by thicker grayish black and grayish white interlacing bands of fibrous tissue. Extending deeply into the lung substance, thickened pleural septa communicated with the fibrous tissue in and about the clusters of tubercles. Except for more pronounced dilatation and thinning of the mucosa, the bronchi of the right lung were similar to those of the left.

The tracheobronchial lymph nodes were moderately enlarged and on section consisted of dense, rubbery, anthracotic centers and thin rims of yellowish white tissue. Calcification was not grossly demonstrable.

The embalmed heart weighed 280 gm. and had the following measurements: tricuspid valve, 120 mm.; pulmonary valve, 80 mm.; mitral valve, 85 mm.; aortic valve, 65 mm.; right ventricle wall, 3 to 8 mm.; left ventricle wall, 15 mm. The greatest transverse cardiac diameter was 13.5 cm. (The estimated normal heart weight on the basis of body length is 317 gm., plus or minus 40.[22]) The tricuspid/aortic valve and pulmonic/aortic valve ratios were 1.84 and 1.23, respectively. (These normally should be 1.68 and 1.05, respectively.[22]) The right ventricle was dilated and its columnae carneae and papillary muscles were more prominent than usual. No mural thrombi were demonstrable and the valvular endocardium was normal.

The enlarged spleen was of normal configuration and measured 18 by 9 by 8 cm. It was covered by a smooth capsule and the splenic

substance was firm and purplish red with normal markings. No tubercles were seen. The liver measured 22 by 16 by 10 cm. The remaining organs showed passive hyperemia and moderate generalized arteriosclerosis.

Microscopic Findings

Throughout the lung there was a conspicuous linear, interlacing, peribronchial and septal pulmonary fibrosis (Fig. 2). This was particularly prominent in the subpleural tissues. The intervening lung tissue was moderately emphysematous. Innumerable tubercles were present in the linear and peribronchial fibrotic areas and were present to a lesser extent in the walls of the respiratory bronchioles and the adjacent alveolar walls. Generally, these tubercles were of two types: sarcoidal and foreign body granulomas. The former predominated by approximately ten to one. Tubercles of these types were intimately associated and, in addition, many intermediate types were presented.

The sarcoidal tubercles (Fig. 4) were free of caseation, contained no demonstrable organisms and were, for the most part, in the same stage of development; however, a minimal number presented some peripheral fibrosis and there was an occasional, coarse, collagenous ball. Generally, the tubercles were sharply demarcated, surrounded by delicate reticulum, and did not present peripheral rims of lymphocytes. They consisted of peripherally arranged epithelioid cells surrounding central, loosely arranged epithelioid and monocytic cells. Giant cells were, for the most part, centrally located and often comprised over half of the bulk of the nodule. The giant cells appeared to be of two types: Langhans' cells and foreign body giant cells, with the former predominating. In many of the Langhans' cells there were numerous small vacuoles, each containing a pink, round body. Other Langhans' cells contained large, clear vacuoles; and, rarely, in those cells containing one large vacuole there was present an "asteroid" body, an intensely eosinophilic stellate mass, 15 to 20 μ in diameter (Fig. 5).

More frequently, the Langhans' cells contained round, oval, or suggestively budding, intracytoplasmic bodies of Schaumann,[1] 25 to 50 μ in diameter. Rarely, these bodies appeared to lie outside of giant cells, and some enclosed irregular yellowish material (Figs. 6 and 7). These bodies stained blue with hematoxylin and in ferrocyanide preparations were strongly positive for iron. Dr. Leroy U. Gardner,[2] who also studied this case, stated that these bodies stained "red with acid fuchsin of van Gieson-Weigert instead of black like elastic tissue" and that "von Kossa's calcium stain is negative." Re-study of appropriately stained sections revealed, as pointed out by Gardner, that the Schaumann bodies did stain red; however, a moderate number also contained calcium in variable degrees, as demonstrated by von Kossa's stain. An occasional giant cell contained one or more clefts suggestive of cholesterol crystals. More frequently, however, doubly refractile, irregular spicules, plaques, and conchoidal masses were observed in giant cells. These doubly refractile masses were often about, or in, the Schaumann bodies, particularly the smaller and partially calcified forms.

The foreign body tubercles were indefinitely demarcated and consisted of rather closely packed, indefinitely arranged, large monocytes, and one or more foreign body giant cells. These tubercles, for the most part, were within the dense zones of fibrosis. Some, however, were present in alveoli and respiratory bronchioles. Golden yellow discoid, verruciform, and incompletely segmented asbestos bodies, many of which were in giant cells, were observed in and about the nodules (Fig. 8). Asbestos bodies, singly or in clusters and in moderate numbers, were present also in the dense fibrotic areas (Fig. 9) and occasionally within alveoli (Fig. 10). Rare, laminated, calcified masses, enclosing apparent asbestos bodies (Fig. 11) and other bodies which appeared to be of the Schaumann variety, were present in the linear fibrotic bands. Asbestos bodies were also encountered in and about the sarcoidal tubercles and in the associated sarcoidal giant cells of both varieties, but more frequently in those of foreign body type. Iron preparations clearly demonstrated the bizarre forms of the asbestos bodies.

In many areas it was difficult to distinguish between the two types of lesions. This was particularly true throughout the subpleural region where both the lesions and asbestos bodies were more numerous, clustered, and embedded in a dense matrix of collagen, masses of coarse elastic fibers, and fine reticulum.

The larger bronchi were remarkable only for slight chronic inflammation. The bronchioles and respiratory bronchioles, embedded in dense collagen and surrounded by tubercles, were moderately dilated and presented conspicuous focal squamous metaplasia and moderate chronic submucosal inflammation. In the subpleural regions where the asbestotic fibrosis and the granulomatous reaction were most intense, the bronchioles were irregularly dilated and lined by alternating strips of tall columnar and squamous epithelium. Only a few bronchioles contained neutrocytic exudate. The respiratory bronchioles were irregularly distended [illegible]

[1] Schaumann, J. On the nature of certain peculiar corpuscles present in tissue of lymphogranulomatosis benigna. *Acta med. Scandinav.*, Vol. 80, 1933.

many contained asbestos bodies and the associated granulomatous reaction. Within the peribronchial fibrous tissue there was a moderate amount of hemosiderin in linearly disposed granules, and fine lipoid droplets. The small pulmonary arteries and arterioles presented slight intimal thickening, and those in the subpleural zone were surrounded by thick collars of elastic fibers.

The intervening alveoli were moderately dilated, the capillaries were congested, and there was a slight increase in collagen in the alveolar walls bordering the fibrous masses. Focally, clusters of alveoli contained lipoid-laden macrophages. "Heart lesion cells" were infrequent.

Sections of the pleura revealed dense, laminated, and oval fenestrated bundles of collagen. Focally, there were indefinitely demarcated nodules which consisted of circularly disposed lamellae of collagen. Superficially, the pleura presented slight fibroblastic activity and an occasional perivascular accumulation of lymphocytes and monocytes, some of the latter occasionally containing hemosiderin. No asbestos bodies were observed. The pleura over the left lower lobe, in addition, bore organizing fibrinous exudate on its visceral aspect.

Sections of the tracheobronchial lymph nodes presented a repetitious pattern of sarcoidal tubercles with almost complete replacement of the lymphoid tissue. Throughout the nodes there were minimal diffuse fibrosis and several nodular masses of coarse collagen. The tubercles were similar to those in the lung as to structure and stage of development. Inclusions of Schaumann were not observed and only a rare "asteroid" was present. Asbestos bodies were not identified. A moderate number of hemosiderin-containing macrophages were present in the remaining lymphoid tissue.

Similar sarcoidal tubercles were present to a slight degree in the spleen and liver, and to a lesser extent in the kidneys, diaphragmatic muscle, and the right and left ventricular myocardium. These sarcoidal tubercles, however, were not as compactly arranged as those in the lung and tracheobronchial lymph nodes, and were surrounded by and permeated by lymphocytes. "Asteroid bodies" and Schaumann bodies were not present in the giant cells of these tubercles. No asbestos bodies were found. Those in the right ventricular myocardium were associated with considerable fibrosis.

The results of chemical and spectrographic analysis of lung tissue, performed under the direction of Dr. Leroy U. Gardner,[26] are presented in Table I.

The final diagnoses were: Moderate pulmonary asbestosis; extensive sarcoidosis of pulmonary and tracheobronchial lymph nodes; marked chronic pulmonary emphysema; slight edema of the lungs; arteries and arterioles in the lungs; marked nodular obliterative pleural fibrosis; focal organizing fibrinous pleuritis; minimal sarcoidosis of the heart, liver, spleen, and kidneys; right ventricular cardiac dilatation and relative right ventricular hypertrophy; acute passive hyperemia of the viscera; slight cirrhosis of the liver; slight generalized arteriosclerosis; minimal focal chronic adrenalitis and nephritis; chronic posterior urethritis and interstitial prostatitis.

TABLE I

Chemical and Spectrographic Analysis of Ash

(Dry Tissue, Approximately 12.4% of Moist Tissue. Ash, 4.00% of Dry Tissue.)

As oxides (except Cl)		As elements		
Chemical analysis			Chemical analysis	Spectrographic analysis
	Per cent		Per cent	Arbitrary units of volume content
Cu, As, Hg, Pb, Bi, Cd, Mo	<0.15	Na	4	
SiO_2	2.76	K	36.7	75
Fe_2O_3	8.03	Sr	None found	100
Al_2O_3	0.37	Be	None found	1.5
BeO	None found	Ca	2.1	5
ZnO	0.38	Al	0.2	80
MnO	0.05	Mg	0.7	50
CaO	2.91	P	8.0	75
MgO	2.18	Si	2.3	60
BaO	None found	Fe	5.6	400
SrO	None found	Mn	0.02	75
TiO_2	None found	Tl	None found	5
V_2O_5	None found	Cu		5
Cr_2O_3	0.07	Ag		25
NiO, CoO	<0.05	Sn		4
Na_2O	5.44	Cr	0.05	3
K_2O	44.40	B		3
P_2O_5	18.42	Be	None found	1
Cl	4.65	Pb		0
CO_2	Present	Zn	0.3	25
		Ni		5
		Pt		10
Total	88.80	Ci	4.6	1
				0

Discussion

Clinically, in view of the significant history of exposure to asbestos, the possibility of sarcoidosis was never entertained. In retrospect, the rapidly progressive, disabling dyspnea, unaccompanied by evidences of enlargement of the right heart or cardiac failure, should have aroused suspicion that there was a concomitant pulmonary lesion. Asbestosis alone is not usually accompanied by such profound, rapidly developing, respiratory embarrassment. In this case, however, there were no collateral clinical evidences of sarcoidosis. It would seem that a clinical diagnosis of coexistent asbestosis and sarcoidosis would be justified only by [illegible]

strate sarcoid lesions and the discovery of asbestos fibers in the sputum, with a history of adequate exposure to asbestos fibers and roentgenographic evidences of diffuse pulmonary fibrosis. Asbestosis of the degree observed, alone should not have caused death, and sarcoidosis has generally been regarded as a benign process. Reisner,[16] however, on the basis of his observations on cases of pulmonary sarcoidosis, stated "that one is not justified in assuming too confident an attitude regarding the ultimate outcome." This statement is particularly true when, as in this case, sarcoidosis complicates pre-existing pulmonary disease.

Pathologically, there were evidences of right heart strain in that there was marked dilatation of the right heart, evidenced by increased tricuspid and pulmonic/aortic valve ratios and slight passive hyperemia of the viscera. The total heart weight, however, on the basis of body length,[23] was normal. As determined by the ratio of the left and right ventricular weights, it has been shown that there may be considerable relative right ventricular hypertrophy without an increase in the total heart weight. However, relative right ventricular cardiac hypertrophy in Higgins' series[16] was not usually accompanied by evidences of right ventricular failure. In view of the significant dilatation of the right side of the heart and the slight sclerosis of the pulmonary arterioles, there was, in all probability, some degree of pulmonary hypertension in this case. However, in the absence of an increase in total heart weight and in the absence of evidences of chronic passive hyperemia of the viscera there was probably no, or insignificant, exaggeration of air hunger due to heart failure.

It has been suggested that dyspnea in the pneumoconioses is due to capillary and arterial blockage by the fibrotic process. This, in all probability, is true to a variable degree in those persons with severe fibrosis of the conglomerate type with attendant extreme chronic emphysema. This hypothesis, however, does not explain the severe dyspnea that is seen in occasional cases of diffuse miliary studding of the framework of the lung by silicotic, tuberculous, sarcoidal, or neoplastic tubercles. It may be that the mechanism of dyspnea in such instances is due to irritation of the vagus nerve endings with reflex stimulation of the respiratory center (Hering-Breuer reflex). In view of the equivocal evidences of hypertrophy of the right heart in this case, mechanical obstruction to the blood flow would not appear to be the responsible factor but, more likely, because of the diffuse active inflammatory process throughout the lungs, the Hering-Breuer reflex was exaggerated. Presumably, there was either a severe respiratory alkalosis or acidosis. Tissue changes suggestive of alkalosis, such as calcification of the renal tubules, were not found.

Microscopically, there was some difficulty in differentiating the two types of tubercles since there were many sarcoidal tubercles which contained asbestos fibers, and tubercles of indeterminate type, not containing fibers or inclusion bodies, were sometimes seen. It was difficult to determine how much of the fibrosis was due to asbestosis. Morphologically, since the majority, by far, of the sarcoidal tubercles were without evidences of fibrosis and apparently of the same age, it is suggested that this process was engrafted upon an established asbestosis. Further, on the basis of Gardner and Cumming's[18] experimental studies on asbestosis, the marked peribronchiolar fibrosis with sequestrated asbestos bodies, the marked pleural fibrosis and pleural septal fibrosis, and the metaplasia of the bronchiolar epithelium indicate that the asbestosis was well established and over 700 to 800 days old. Dr. Leroy U. Gardner, who kindly examined the material, stated: "In comparison with our other material the pigmented foci in your case seem to show more fibrosis and less localized emphysema. Histologically, this can probably be explained by the presence of sarcoid nodules within the asbestotic zones of reaction. I would infer that in your case the two conditions developed more or less simultaneously, but that probably the asbestosis was present to some degree before the sarcoid appeared. This opinion is based upon the occurrence of asbestos fibers and other iron-containing particles in the interior of the tubercle-like nodules and in some cases within the giant cells themselves. The number of asbestos bodies is smaller than seen in many cases."

Inclusions of the Schaumann variety, found only in the lung, occurred in 4 per cent of the giant cells. Some of these enclosed golden-yellow, irregular bodies suggesting asbestos bodies, but similar to organic material previously described within such bodies. Yet there were definite asbestos bodies enclosed by similar dark blue material. Schaumann inclusions have been described in only 4 per cent of the reported necropsies on sarcoidosis as summarized by Rubin and Pinner,[17] who did not regard these inclusions as specific for sarcoidosis. Rich,[22] who was impressed by the frequency of Schaumann inclusions in sarcoidal lesions and by their absence in unequivocal tuberculous lesions, noted that Metchnikoff reported the presence of calcified inclusions in the hyperplastic tuberculous lesions of experimentally infected Algerian rats. Kraus[20] stated that the presence of calcified inclusions was a feature not found in any known granuloma except

sarcoidosis. Gardner[24] pointed out that, in his sarcoid material, these bodies, regarded by many to consist of calcium or calcified remnants of elastica, do not, by the von Kossa method, contain calcium, but, by the ferrocyanide method, give a strong reaction for iron. Only a moderate number of the Schaumann bodies observed in the present case were either wholly or partially calcified, yet all gave a strong reaction for iron. Studies on sarcoid lesions of lymph nodes and spleen from another case revealed only a few iron-staining noncalcified Schaumann bodies. The presence of doubly refractile, nonlipoid substance in giant cells and frequently in close relation to Schaumann bodies has not been emphasized in the literature on sarcoidosis. It has been noted, however, that colorless and yellowish tinged refractile material is often enclosed by the Schaumann body. The fact that these masses are frequently doubly refractile has not been stressed. It has been suggested that these enclosed masses represent disintegrating elastica; however, van Gieson-Weigert stains do not confirm this suggestion. The origin of this refractile and doubly refractile material is not known. Being in and about many of the small, partially calcified bodies, this doubly refractile material appears to be associated with the development of the Schaumann body. The larger and more densely stained bodies were not as frequently associated with visible doubly refractile substance. However, fractured and fragmented, apparently old, Schaumann bodies, as seen in control sarcoid material from lymph node and spleen, usually contained moderate amounts of doubly refractile substance. Apparently then, the Schaumann body, which stains blue with hematoxylin and red with acid fuchsin, is formed in response to doubly refractile, nonlipoid substance and initially is impregnated by iron and later, in amounts demonstrable by von Kossa's stain, by calcium.

Wolbach,[21] in 1911, Jadassohn, in 1919,[25] and Friedman,[18] in 1944, have described a peculiar intracellular body in cases of sarcoidosis. This body, stellate in shape, varies in size up to 25 μ, generally lies in an intracytoplasmic giant cell vacuole, and stains intensely with acidophilic stains except the central area which is basophilic. Wolbach described them as lying free in tissue spaces, in endothelial leukocytes, and in giant cells. Friedman found such bodies in only 6 to 8 per cent of the giant cells in his case. Both investigators attempted to determine the chemical structure of this stellate body by specific stains; however, they were unsuccessful. Both considered the possibility of its being an extraneous organism, although questionable. Wolbach regarded it as a nonspecific biochemical alteration of the cytoplasm. He was never able to demonstrate stellate bodies in other material and decided that they were not similar to inclusions sometimes seen in cases of sarcoma. Friedman regarded these bodies as nonspecific but highly characteristic of sarcoid lesions. Friedman proposed that these bodies be called "asteroids," but perhaps it would be better, eponymically, to call them Wolbach's asteroids. They have been described in 7 cases of sarcoidosis, and never in association with the Schaumann calcified inclusion body. In the present case asteroids were present in approximately 1 per cent of the giant cells in the lungs and tracheobronchial lymph nodes. Definite transition stages of asteroid formation were suggested by the presence of spicules on the pink, coccoid, intravacuolar, intracytoplasmic bodies, particularly in those giant cells in which the small vacuoles were clustered and disintegrating. In addition, an occasional Wolbach's asteroid, instead of lying in a large, clear vacuole, was surrounded by agminated ruptured vacuoles. In view of the presence of similar pink, coccoid, intravacuolar bodies, similar asteroids, and the same suggestive stages of asteroid formation in the giant cells of talcum powder granuloma, as observed in one case in this laboratory, these giant cell cytoplasmic changes must be regarded as Wolbach originally suggested, nonspecific biochemical cytoplasmic alterations. In addition, such an asteroid is depicted in the giant cells of leprous lesions by Mallory[26] who called them "spiculated" bodies. No transition stages between Wolbach's asteroids and Schaumann's inclusions were even remotely suggested.

The pathogenetic relationships of asbestosis and sarcoidosis are dependent upon the chronologic development of the lesions and the nature of the causative agents. Historically and histologically, in this case, it is most likely that asbestosis preceded the development of sarcoidosis. The predominant localization of the sarcoidal tubercles within asbestotic zones of fibrosis with attendant morphologic modification of both lesions, as evidenced by asbestos bodies within sarcoidal tubercles and lesions of indeterminate type, would suggest an analogy to the intimate relationship existent between tuberculosis and the pneumoconioses. If must be remembered, though, that even in uncomplicated sarcoidosis the lesions occur in the framework of the lung, and therefore the morphologic relationships of the two may be coincidental. This would be in agreement with those who believe that morphologically sarcoid is not reconcilable with tuberculosis. However, to these who regard sarcoidosis as a peculiar form of tuberculosis, this case then would be one of asbestosis with superimposed noncaseating tuberculosis.

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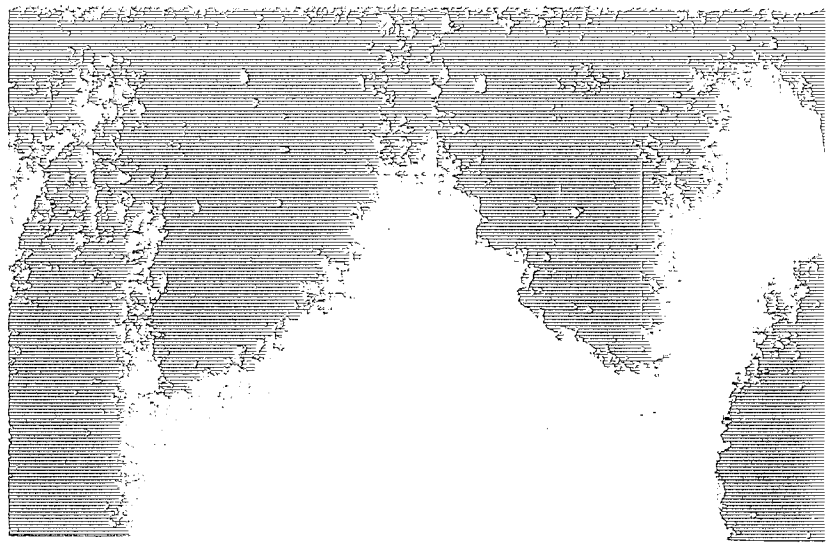
[*Illustrations follow*]

DESCRIPTION OF PLATES

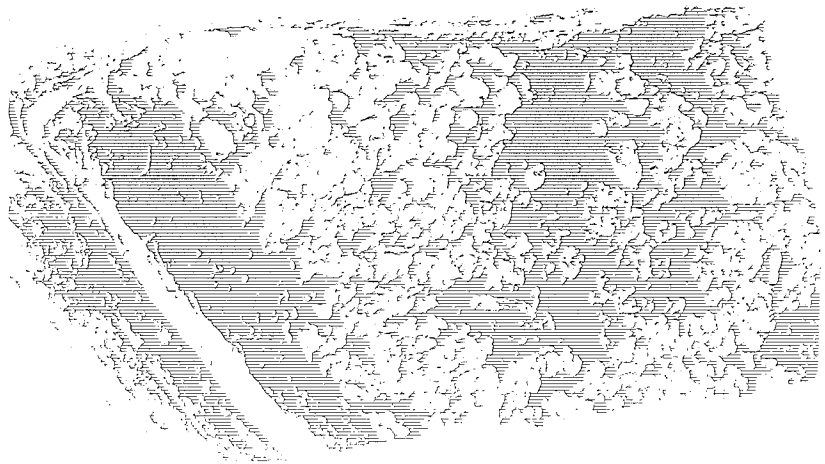
PLATE 103

FIG. 1. Initial roentgenogram of the chest.

FIG. 2. Photomicrograph of lung and adherent pleura showing subpleural, septal, peribronchiolar, and marked and focally nodular pleural fibrosis. × 4.



1



2

PLATE 123

FIG. 3. Frontal section of left lung.

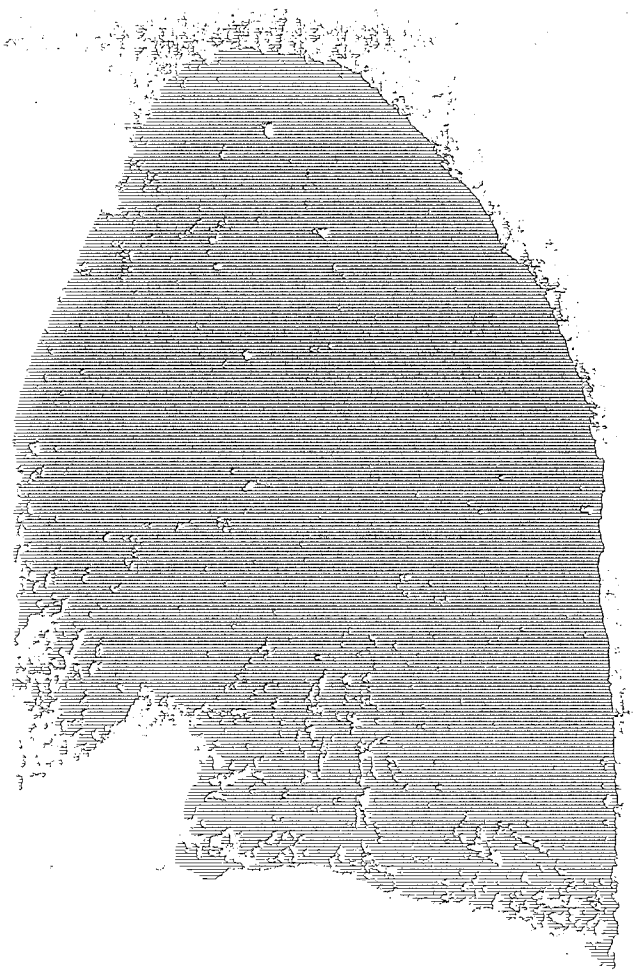
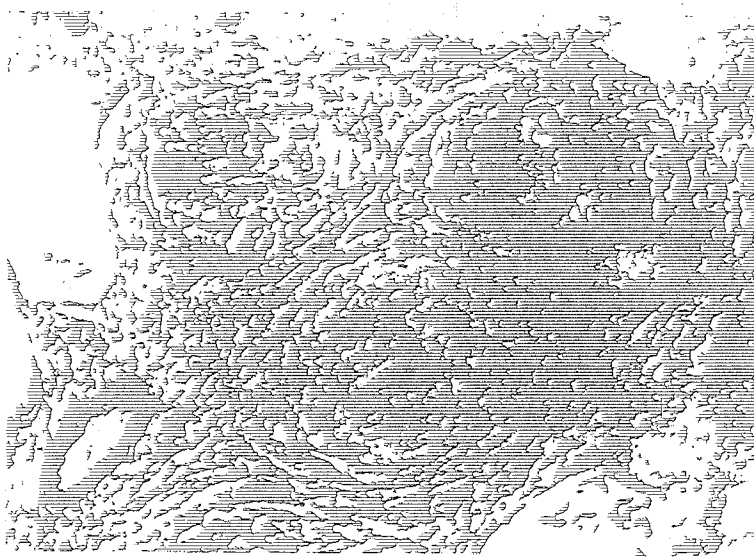


PLATE 105

FIG. 4. Lung, showing a cluster of sarcoidal tubercles. × 150.

FIG. 5. Lung. The giant cell which nearly fills the field contains an "asteroid" in an intracytoplasmic vacuole. × 1090.



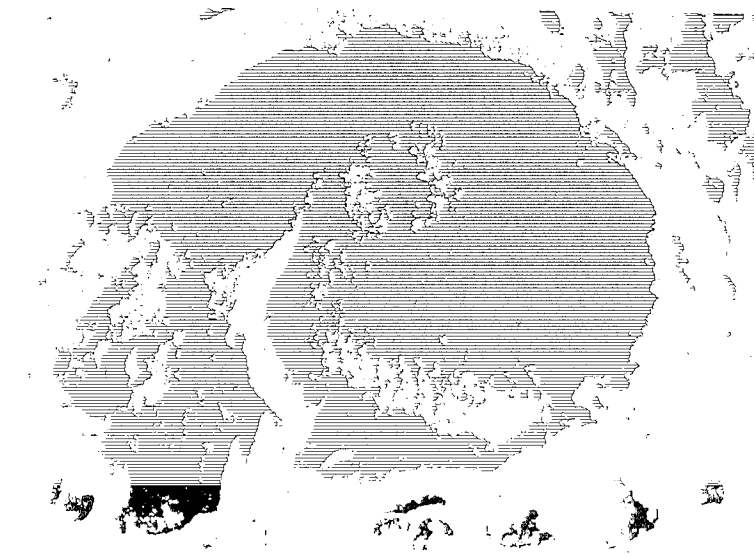
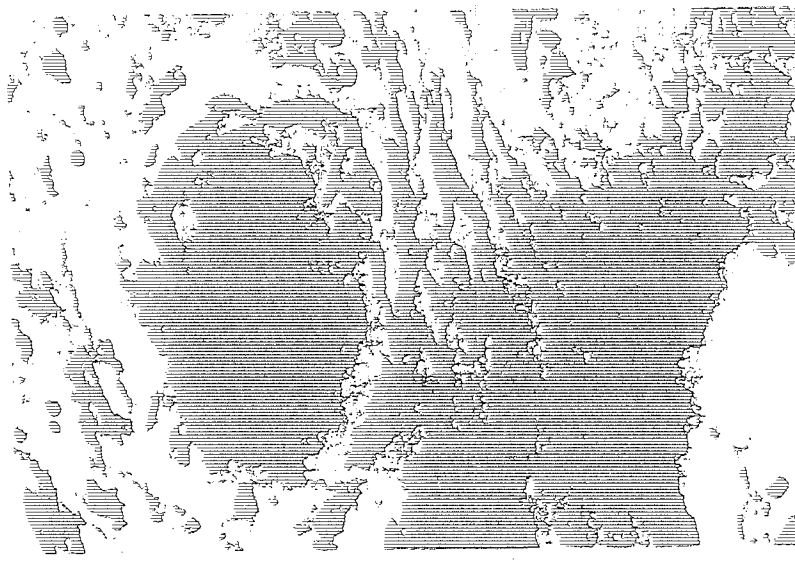


PLATE 106

FIG. 6. Lung. An inclusion of Schaumann encloses an oval yellow body. With polarized light, doubly refractile material surrounds this calcified mass. × 725.

FIG. 7. A giant cell from the lung with inclusions of Schaumann. × 725.



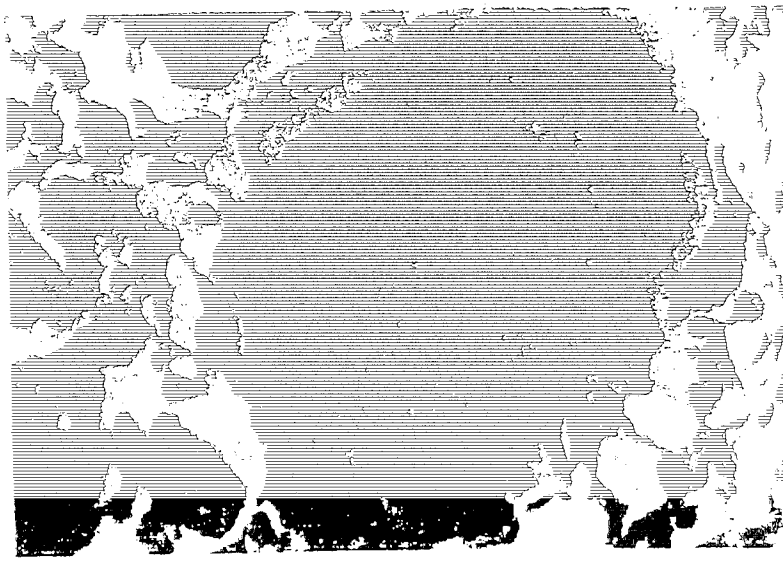
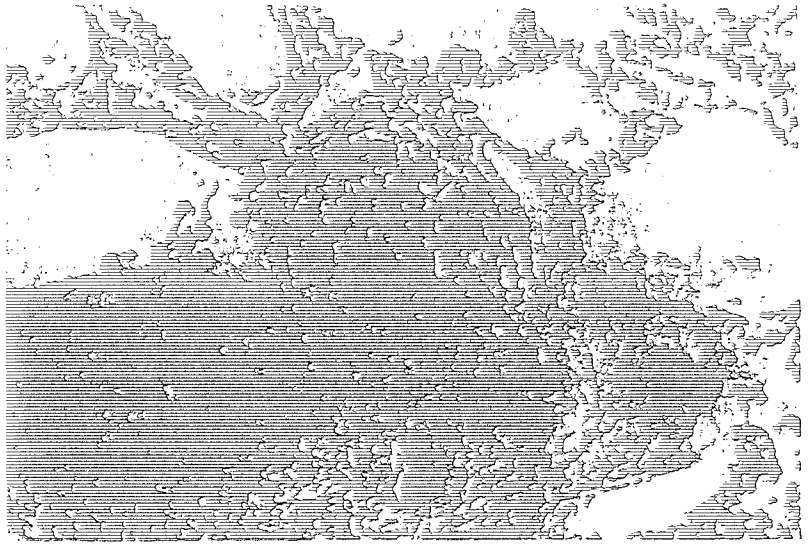


PLATE 107

FIG. 8. Lung with tubercles of foreign body type. The central tubercle has an asbestos body at its periphery. × 160.

FIG. 9. Lung showing asbestos bodies and clusters of hemosiderin-laden macrophages within an area of fibrosis. × 725.



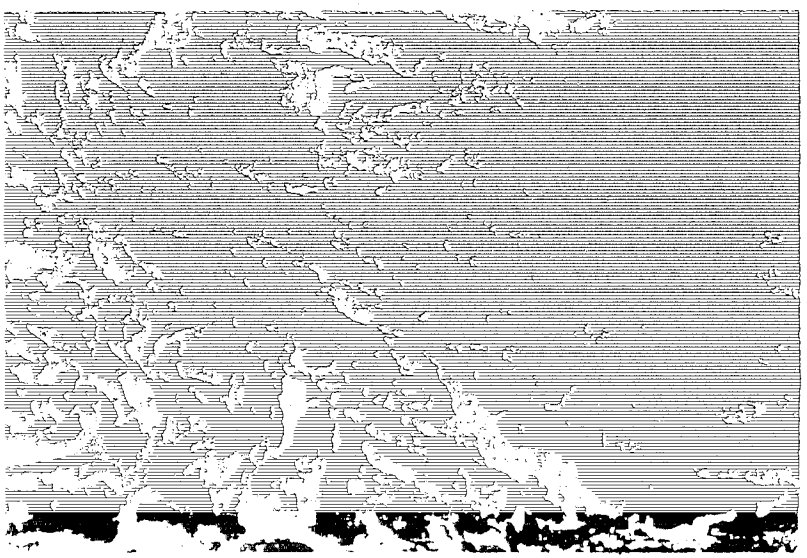
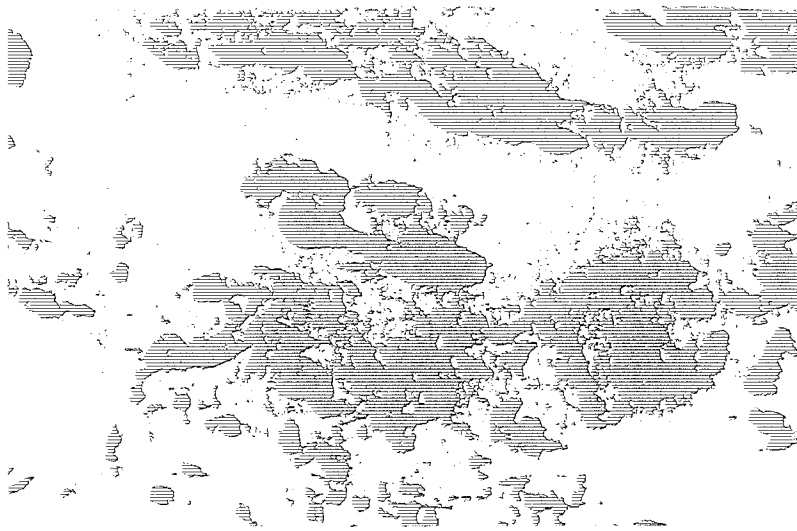


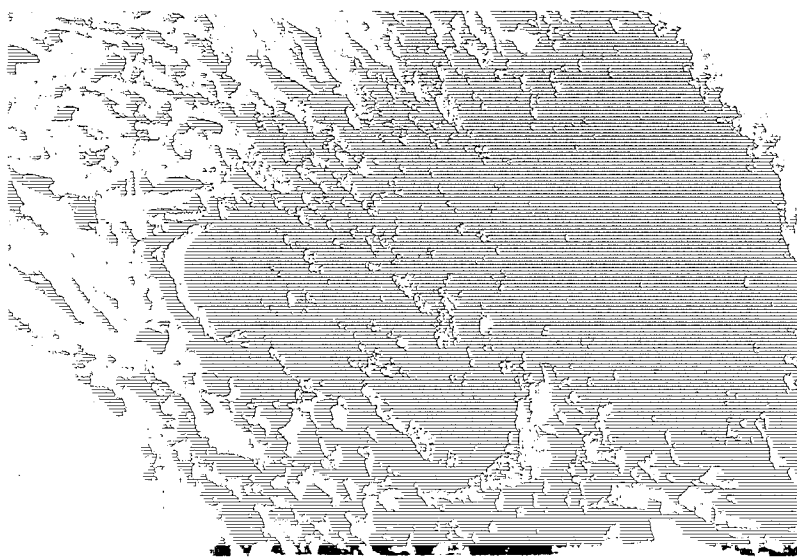
PLATE 108

FIG. 10. An asbestos body is shown in an alveolus. × 725.

FIG. 11. Lung showing an asbestos body encrusted with iron and calcium. × 725.



10



11

County of Westchester

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THE HONORABLE EL EL
JOSEPH E. CASEY
CLERK

COUNTY COMMISSIONERS
E. L. CAMPBELL
MRS. F. WOOD
JOSEPH L. WHEELER

October 1, 1954

Robert Levy C. Gates
Director of Hospital Laboratory
Garnet Lake, New York

Dear Bobby Gardner:

Hello, I am writing to you for an opinion. This time in regard to a case thought to be one of tuberculosis. Dr. Davies and I really appreciate it if you could find time to review the case.

The chest findings are, I believe, consistent with published reports. However, stereoscopically, numerous active and regressive tubercular lesions are encountered in the lungs and the tracheo-bronchial lymph nodes and a rare tubercle is found in the spleen, liver, pancreas and kidney. The character and distribution of these non-caseating tubercles is suggestive of sarcoidosis. Yet, in the lung nodules are present which some of the lesions, and within foreign body giant cells. In addition, there are areas of linear fibrosis with lesser calcified bodies.

The paucity of calcified bodies, as compared with sections yet lately must to be several years ago, the absence of a bare-processed fibrosis in the lungs, and the tuberculous lesions in the lungs, nodes, and several of the spleen, have led me to believe that the lesions are mixed, being one predominantly of sarcoidosis, and minimally one of tuberculosis.

Now is, to my knowledge, the first necropsy in which tuberculoid bodies have been demonstrated in one series or that of the Riverside General Hospital. In view of this and, to me, the peculiarity of the lesions it would be extremely desirable to have your expert opinion.

I am sending under separate cover, stained and unstained slides, the necropsy report, and clinical abstract. The limited occupational history in all we have at the present time.

Very truly yours,

R. J. Ritterhoff, M. D.
Director of Laboratories

PLAINTIFF'S EXHIBIT #1
R. Ritterhoff

November 10, 1944

Dr. E. J. Lesemann
Director of Laboratories
County of Los Angeles
Los Angeles General Hospital
Post Box
Cincinnati 9, Ohio

Dear Dr. Lesemann:

I am most grateful to you for allowing me to see the sections on your unusually interesting case of sarcoma and the report of the lungs. I agree that this combination is one of the rarest of the rare as far as I know and that it would be most desirable to publish your findings.

Obviously the cancer constituted the predominant pathological change and there is in addition a dilated aspect of the bronchi. It is hard to be sure exactly how much of the bronchial dilatation is due to the marked cough. However, this is a white extensive an you probably and the number of cancerous bodies is quite seen in many cases. One would infer that the cancer's exposure to cancerous fibers had been limited although the occupational history says quite about the nature of his work.

I hope I may be granted to keep these slides as they are really very beautiful.

It gives me to have a chance study to determine the amount of microscopic growth in the lungs. The of cancerous cells on the slides is extremely helpful to me in interpretation. Thank you, I am so glad to cooperate. Again may I thank you for allowing me to see the material.

Sincerely yours,

Leroy U. Gardner, M. D.
Director

LUG:MB

PLAINTIFF'S EXHIBIT #2
Ritterhoff

Board of Trustees
[illegible]
[illegible]
[illegible]
[illegible]

Church of Salvation

THE TUBERCULOSIS HOSPITAL
PRICE HILL
CINCINNATI, O. OHIO

[illegible]
[illegible]
[illegible]

Staff Consultants
[illegible]
[illegible]
[illegible]

December 23, 1944

Dr. Leroy U. Gardner
The Gamma Laboratory
Geneva Lake, New York

Dear Dr. Gardner:

I am very grateful to you for rendering my case of carcinoma and osteomyelitis. Needless to say, your confirmation of the diagnosis, even though I was prone as to the degree of suboccipital pressure, made me very happy—gives produced a few "chuckles." Your suggestions as to penicillin will be followed; I would like permission to quote you and include your findings in this report.

Plans is being made for chemical analysis. Would it, also, be advisable to have x-ray diffraction studies for silica? Unfortunately, I can unable to give you supplemental data in regard to his occupational history, as the local factory concerned has been uncooperative. They have not permitted me to visit the plant, nor have they given in detail his occupational history.

I have several questions concerning phases of this case about which I would like your opinion:

First, in some of the giant cells and occasionally within areas of fibrosis there are rare clefts encrusted subacute bodies. Have these been previously described?

Second, would you care to comment upon the possible etiologic and chronologic relationships of these two diseases. I note in your letter, that pathologically "it is hard to be sure how much of the fibrosis is due to inhaled dust." Is it probable that carcinoma antedated and led to inhaled dust? Or is it probable that carcinoma is a modified tuberculous infection and you believe that carcinoma is a modified tuberculous infection and you

PLAINTIFF'S EXHIBIT #3
Ritterhoff

County of Hamilton

THE TUBERCULOSIS HOSPITAL
PRICE HILL
CINCINNATI, 5, OHIO

WM. B. BRODERICK, M. D.
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report, Jour. of Indus. Hygiene Vol 13, March 1931, on experimentally produced tuberculosis in asbestos dusted cavies, I favor the belief that in this case it is likely that asbestosis predisposed to tuberculosis.

I will greatly appreciate your help in regard to chemical analyses, and your comments, if this permits, to the above questions.

Yours truly,

E. J. Witherby, M. D.
DIRECTOR OF LABORATORIES

EJW/bmp

December 27, 1944

Plaintiff's Exhibit #4
Ritterhoff

P. N. G. Klebsiella
Director of Laboratories
The Tuberculosis Hospital
Pilot Butte
Saskatchewan, Ohio

Dear Dr. Klebsiella:

I am very pleased indeed that you could let us have such a generous sample of blood for checking serologic of your Pneumatinga case of carcoid at Sabeatooth. We will give it the very best attention to ascertain what are present. The story with Indian City Hospital and X-ray difficulties analyses but I shall send us out for a photomicrographable analysis. I am doing this because I am looking for control material to a series of carcoid of carcoid-like cases from the Tuscaroora Zero Laundry. I am doing everything that I can to ascertain whether there may be an industrial environmental factor involved in the etiology. Thus far we usually find very little in these people save Aureomycin and other types of leprosy. Sorrilla is under complete bib in spite of intensive study the ultrasound to any petroleum ether-ether ether vapor Plates. Experimental work you may of the microcella Lavaford has so far progressed to visible results.

It is my opinion that we could do probably two things which seem to be useful at the present time. Perhaps it is true. I don't remember the Klebsiella plates with Pneumatinga A Pinus Lobe so easily for Coon it was Lucy that the disease is due to Lepra bacilli or other acid-fast organisms. I believe that it may be Corynebacterium Lepraemurium, its role as a Gram. pro- duly stain as a Negative organism so also the virus was found to do it as well.

In your case the presence of carcinoma could seem to be probably entirely coincidental, as I have said to the first time the combination has been reported. I have studied on approximately five cases of carcinoma and I have never seen one complicated by carcinoma. I would like to in your case the two conditions developed more or less simultaneously but that probably the tuberculosis was present to some degree before the carcoid appeared. The opinion is based on the presence of numerous fibrous and other Lung-containing particles in the lacunae of the tuberculo-like nodules and in some cases within the Giant cells themselves. The calcium encrusted inclusions in the plant cells are very frequent in all of the pulmonary carcinoma that I have studied. I do not know what they are but they look like bits of glass

Glass that have attracted your calls; others have an oyster shell appearance whose origin is hard to imagine. I see in the sections of your cane a few of those dooply stained bodies that are a feature of carcoid and in addition, I guess, as you mentioned, certain porcelain of fibre that have a yellowish cast which can suggested may be asbestos. Those, of course, are not intended to unnecessarily complicate. On the other hand, some of the carbonate bodies in preparations have caused real disarray apparently.

As the idea of being cautious about the commercial aspects I would appreciate it if you would tell me what sort of a plan the Philip Carey Manufacturing Company operates and whether you have any idea of the nature of their products. I am interested, of course, but only in the exposure to asbestos but in the possibility of an exposure to fibrous substances. We shall probably be able to determine the presence or absence of the latter by electronic analysis but it would be helpful to know what possibilities are involved.

Personally I would hesitate to comment greatly on the question of causal relationship between asbestosis and carcoid. Such relationship may exist but from the evidence before us I would find it difficult to decide which was cause and which was effect. In view of the fact that the two conditions occur so rarely in the same lung I would rather entertain the opinion that chance was responsible for this simultaneous occurrence in your case.

After we get our various chemical and other analytical data together I would be very glad to give you my further opinion to be used in your case report, if you so desire. I will defer that action respecting the gross material I can secure together with the sheets of asbestosis. In cooperation with our other material the pigmented tool in your case does to show more fibrosis and less localized emphysema. Also- logically this can probably be explained by the presence of carcoid nodules within the asbestotic zones of fibrosis.

Again may I express my appreciation for being allowed to study this unusually interesting case.

Sincerely yours,

Leroy U. Gardner, M. D.
Director

LUG:PE

PLAINTIFF'S
EXHIBIT
#5

characteristic peribronchiolar relationships are no longer recognizable.

The sarcoid lesions are small fibrous or more rarely cellular nodules containing one or more giant cells. As in uncomplicated cases they occur in interstitial tissue, the sheaths of bronchi and blood vessels and the alveolar and interlobular septa. In addition, nodules are found at the margins of areas of the dust fibrosis. Where the dust reaction is heaviest, especially beneath the pleura, the nodules are definitely within the asbestotic fibrous tissue.

The giant and epithelioid cells of the sarcoid show no evidence of degeneration as evidenced by fat stainable with Scharlach R. They are surrounded by a delicate reticulum (black with Foot-Bielschowski) and at the periphery of the nodule are concentric bands of heavy non-hyaline collagen. The elastic tissue is probably pushed aside in the formation of nodules.

Inclusion bodies in the giant cells of the sarcoid granulomas are fairly numerous; those that occur are largely of the round or oval creased conchoidal type. These stain black with hematoxylin, red with the acid fuchsin of Van Gieson-Weigert instead of black like elastic tissue. Von Kossa's calcium stain is negative. <u>They give a strong Prussian Blue reaction for iron.</u> On testing other cases of sarcoid it is found that in them also the inclusions contain iron and not elastic or calcium. Occasionally typical asbestosis bodies are found within the sarcoid nodules.

The asbestosis bodies in the pneumoconiotic tissue reveal marked and unusual modification in ferrocyanide preparations. Many of the bodies have one or several very large swellings either terminal or in the middle of the fibre. Bizarre forms develop about the partially separated bundles of asbestos. The conchoidal rounded masses are much more abundant here than within the sarcoid nodules. Much of the granular dust in the lesions is apparently iron.

The blood vessels and bronchial tree show nothing unusual except for the sarcoid nodules in their sheaths.

There is no evidence of inflammatory reaction or pneumonic exudate.

<u>LYMPH NODES</u> - Largely replaced by sarcoid nodules varying from cellular to fibrous in character. No necrosis. All show giant cells vacuolated but no inclusion bodies. Varying numbers of lymphocytes in most of the lesions.

<u>SPLEEN</u> - A few small sarcoid nodules with giant cells but no inclusion bodies.

<u>LIVER</u> - Same as spleen.

<u>KIDNEYS</u> - 0 sarcoid; small foci of schlerosis with arteriolar sclerosis.

CHEMICAL AND SPECTROGRAPHIC ANALYSES

Ash 6.70% of Dry Tissue.
(Dry Tissue approximately 14.1% of Moist Tissue)

Ash

Cu) As) Hg) Pb) Bi) Cd) Mo)	Less than 0.15%
SiO_2	2.76
Fe_2O_3	8.03
Al_2O_3	0.37
BeO	None found
ZnO	0.39
MnO	0.03
CaO	2.92
MgO	1.18
BaO	None found
SrO	" "
TiO_2	" "
V_2O_5	" "
Cr_2O_3	0.07
NiO) CoO)	Less than 0.05
Na_2O	6.44
K_2O	44.40
P_2O_5	18.42
Cl	4.61
SO_3	——
CO_2	Present
	88.82

Elements

	From Chemical	Spectrographic
Na	4	75
K	36.7	100
Sr	N.F.	1.6
Ba	N.F.	5
Ca	2.1	80
Al	0.2	50
Mg	0.7	75
P	8.0	60
Si	1.3	100
Fe	5.6	75
Mn	0.02	5
Ti	N.F.	5
Cu		25
Ag		2
Sn		3
Cr	0.05	3
B		1
Be	N.F.	0
Pb		25
Zn	0.3	5
Bi		10
Mo		5
Cl	4.6	0