ESB INCORPORATED

(FORMERLY THE ELECTRIC STORAGE BATTERY COMPANY)

EXECUTIVE OFFICES

2 PENN CENTER PLAZA, PHILADELPHIA, PENNSYLVANIA
(215) LOCUST 4-4030

MAIL: P.O. BOX 8109, PHILADELPHIA, PA. 19101
CABLE ADDRESS: ESBCO PHILADELPHIA

September 27, 1968

R. A. Kehoe, M.D.
Kettering Laboratory
College of Medicine
Cincinnati, Ohio 45219

Dear Dr. Kehoe:

I think it has been a number of years since you were in any way associated with The Electric Storage Battery Company. In these years we have changed the name of the company as you can see, but one of our major problems is still lead exposure. We no longer see any of the symptoms which were considered almost inevitable in the old days, but we do continue to get an occasional claim. We have made a few compromise settlements, but we find now that the time has come to make a stand and, also, to ask you to work with us again.

Please let us know whether you are doing consulting work of this type and if you would consider testifying in a workmen's compensation hearing in Allentown, Pa. The time of the hearing would probably be no earlier than November of this year.

I am enclosing some information on the claimant to assist your in making a decision. We feel that the claim is a weak one and that the possibility of lead being the cause of this man's health problems is remote. We would be pleased to go into all aspects of the case and the plant's lead control procedures with you in detail.

Sincerely,

W. M. Pallies
Health and Safety Manager

WMP/sm
enc.

N 6152

FRANCIS S. KLECKNER, M.D.
202 NORTH EIGHTH STREET
ALLENTOWN, PENNSYLVANIA 18102

Area Code 215
Telephone 432-5001

March 22, 1968

[redacted]

AH # 511511
Admission: 2/12/68
Discharge: 3/2/68

Admission diagnosis: Abdominal pain, unknown etiology

Discharge diagnosis: Plumbism
Cholestatic hepatitis, unknown etiology

Surgical proceedures: Liver biopsy (needle) 2/22/68 and 2/29/68

This 35 year old white male had been admitted to the Allentown Hospital in January 1968 for recurrent abdominal pain and laparatomy which revealed no abnormal finding and was discharged on January 11, 1968 with the diagnosis of suspected pylorospasm and underlying gastritis. Follow upp care was by Trexler Medical Group and with recurrence of his abdominal pain, the patient was scheduled for G.I. consultation at my office. On 2/12/68 the patient had a severe onset of the same type of upper abdominal pain which had become progressively worse over the previous three weeks. The pain had been nocturnal was not relieved by food or antacid, was not altered by position, it, in fact was not controlled on a bland diet, antiacids, anticholenergics therapy. The pain was quite severe causing anorexia and nausea but no vomiting. There were no signs of hematopesis, melena or hematochezia. The patient denies the presence of jaundice, brown urine, or grey colored stools.

The past history of this patient is most significant that he was admitted to the Allentown Hospital in 1950 for abdominal pain similar in nature and at this time the red blood cells were found to be stippled and a diagnosis of lead poisoning was made by the attending physican. However in review of the chart on microfilm, no other lab evaluation of significance to this diagnosis were present. The patient had been employed in 1950 by the Caloric Company in Topton, Pa. and had worked for them for 3 months however to his memory he was told that they were not using lead based paint. For the past 12 years the patient has been employed at various positions by the Willard Battery Co. of Allentown, Pa. and to his memory has had elevated urine levels (probably measurements of coproporphyrins) and in fact, because of these evevated levels was moved to a low from a high lead levels area about one year ago by the company. I had asked Dr. Gernard to forward to me the patient's blood and urine levels reflecting lead absorption but to this date, the information has not been received, Dr. Gernard is the company physican.

There is no family history of metal poisoning either in the patients present family, his parents or grandparents. Neither is there a history of liver disease, blood disease, or kidney disease. Father died at age 49 of TB, mother age 60 is alive having sustained a heart attack.

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ell-developed , white male weighing 145 lbs, height 5'7",
it abnormalities were tender right upper quadrant of the abdomen with normal bowel sounds , question of rebound tenderness in the right abdomen , right flank tenderness, . There was no descernable hepatomegaly and splenomegaly was absent as were other masses in the abdomen. Rectal exam revealed brown stool and tested in the dispensary it was guaiac negative. Initial impression was plumdism vs. pancreatitis vs porphyria vs ulcer.

Lab. Evaluation: Pre admission 2/9/68 hemoglobin 15.8, hematocrit 46, WBC 6800, 54 segs , 44 lymphs, 2 eosin, serum amalyse of 65 units. On the day of admission 2/12/68 urinalysis SG 1.010, PH 6.5, negative for protein and sugar, normal microscopic. Serum amlyse 65 units, hemaglobin 16.2, hematocrit 48, WBC 8000, 55 segs, 43 lymphs, 2 eosins. 2/13/68 blood lead level .079 mgms % or 79 mcg %, the normal being 20 mcg %, urine coproporphyrin slightly elevated, uroporphyrin normal. 4 hour urine amalyse 384 units in 295 cc of urine, serum bilirubin total 1.3 mgms%, direct 0.4 , RBC morphology was negative for stippling, serum protein electrophoretic pattern on blood drawn 2/13/68 revealed a normal pattern total protein of 7.6gm %, albumin of 4.27 gms % on 2/15/68 amino defussion of the globlin revealed IGG 1000 normal 8 80 to 1500, IGA 360 normal 165 to 409, IGM 145 normal 50 to 110, all in mgms %, the interpretation therefore was normal. On 2/13/68 the BUN 11, uric acid 5.7, creatinine 0.8, FBS 76, serum electrolytes were sodium 148, K 4.7, CL 113, C)2 26 meq/liter, alkaline phosphatase 5.6 BU. On 2/15/68 the urine coproporphyrin was moderately elevated , the uroporphyrin was normal. The porphobilinogen was negative. Urinalysis: SG 1.015, PH 5.5, negative for protein and sugar, normal microscopic and when I tested the urine the urine contained bile. Serum bilirubin was 4.2 mgms% total with 1.2 direct. On 2/16/68 RBC indocins MCV 90, MCHV 29.5, MCHC 33.5, hemoglobin 16.6, hematocrit 50, RBC 5.5 million, lee hite 6 minutes, PT time 100%, BSP 26 % retention at 45 minutes , reticulocyte count 0.4 %. This day the urine for porphrobilinogen was negative and stools 2X were negative for guaiac. On 2/16/68 SGOT 150, SGPT 180, 24 hour urine contain 3.5erlic units , normal 1-15, 24 hour urine from 2/18/68 to 2/19/68 of 1930 cc contained corphyrins 470 mgms , normal 00160, uroporphyrin 72 mcgms normal 0-26. 2/19/68 the alkaline phospotase was 11.9 BU, hemoglobin 14.2, hematocrit 43%, total serum bilirubin 1.4 mgms %, with direct of 0.4, SGOT 200, SGPT 290. 2/21/68 alkaline phosphatase 12.8 BU, calcium 10.4, phosphorous 3.5, total bilirubin 1.6 mgms, direct 0.7, SGOT 174, SGPT 120, calcium versenate therapy was started on 2/23/68. On 26/68 hemoblobin 15.6, hematocrit 45, WBC 4500, segs 49, lymphs 47, mono 3, eosin 1, calcium 10, phosphorous 4.2 mgms, alkaline phosphatase 17.4, normal serum electrolyes, tootal bilirubin 1.6 mgms %, SGOT 142, mono test negative. 2/27/68 Urinalysis : SG 1.006, PH 7, negative for protein and sugar, normal microscopic, negative for bile in the urine. 2/29/68 hemoglbin 13.8, hematocrit 41, blood lead level 47 mcgms %, 3/1/68 urine corprophyrins 214 mcgms, uroporphyrins 47 mcgms , in a volume of 2000 cc, these studies done by bio-Science Labs. 3/2/68 alkaline phosphatse 8.6 BU, SGOT 44, SGPT 104, total bilirubin 1.3, direct 0.5 mgms %, hemoglobin 15.2, hematocrit 44, WBC 5400, normal differential. Gastric analysis done 2/15/68 revealed basal collection 61cc 0.05 meq of acid, however the presence of blood invalided this value, histalog stimulation one hour collection of 130cc contained 18.2 meq of acid . Impression:hypersecretory ability . Special hematology did a hemoglobin , electophoresis with a value of 135 mgms % - normal.

X-Rays (C32019) 2/13/68 abdomen and chest x-rays , no abnormal findings. Upper G.I. series , no abnormal findings but the exam was incomplete. Upper G.I. series 2/16/68 Dr. Hunter impression was that there was an ulcer 4 mm in diameter at the base of the duodenal bulb, the junction between the pylorous and the duodenum. Personal impression is that this is not an ulcer defect but rather the entrance

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of the pyloric channel into the duodenal bulb , and I point out that there is no irritability of the duodenal bulb or the pylorus , neither are there classical signs of radiation folds coming from this "ulcer" . Personal impression: this is a normal duodenal bulb and that no active ulcer is present. 2/20/68 barium enema normal, 2/24/68 cholecytogram 3 successive attempts to pacify the gall bladder have been unsuccessful, the same visualization is seen. 2/26/68 the fourth day of exam following injection of Orografin does not reveal any stones nor does it reveal any concentration of dye within the gall bladder. 2/26/68 liver scan normal. Pathology # 68-1040 2 liver biopsies revealed normal liver histology .

Consultations: Hematology consultation Dr. David Prager obtained on 2/23/68 his impression was lead intoxication, possible hepatitis due to viral infectious mononuclealous, hepatitis or toxic hepatitis, he recommended a mono test and liver scan if necessary , did not detect any evidnce of lead intoxication relative to the red blood cells as a account for the hyperbilirubemia.

Hospital course: the hospital course was highlighted by recurrence of abdominal pain relieved by Demerol injections . On 2/15/68 while the patient was having a gastric analysis we collected a urine which was brown (dark) in color, tested it and found bile present and in addition found coprophyrin present by the fluorescent method. Thus we considered the abdominal pain as related to either to porphyric or lead poisoning. With the finding of porphrobilinogen absent on 2 occasions, with elevated coproporphyrin and blood lead level and with continuation of the patient's abdominal pain having relief only with demerol there seemed to be little question this man did not have porphyria and did not have ulcer disease. He was started on a course of calcium versenate therapy 500 mgms every 12 hours for 5 days and from that time forward was asymptomatic . Two liver biopsies were normal and the cholestatic jaundice the patient had remitted. The patient was discharged 3/2/68 free of pain, weighing 139 lbs. He will be followed by the Trexlertown Medical Group and myself.

Francis S. Kleckner, M.D.

CC Allentown Hospital
Trexlertown Medical Group
Union Representative

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Date	Urine		Blood
	Specific Gravity	Mg Pb/liter	Mg Pb/100 gm blood
11-17-63			0.063
01-20-64	1.020	0.18	
08-12-64	1.025	0.15	
11-10-64	1.022	0.15	
12-18-64	1.023	0.16	
07-07-65	1.018	0.18	
08-03-65	1.018	0.08	
09-01-65	1.011	0.11	
10-06-65	1.016	0.26	
10-21-65			0.105
11-02-65	1.017	0.21	0.062
12-02-65	1.020	0.39	
12-16-65			0.092
12-30-65			0.097
01-07-66	1.015	0.07	
01-25-66			0.063
02-07-66	1.024	0.18	
02-08-66			0.085
02-15-66			0.059
02-28-66			0.061
03-07-66	1.020	0.26	
05-05-66	1.020	0.15	
06-03-66	1.024	0.26	
06-23-66			0.093
07-05-66	1.032	0.26	0.077
08-02-66			0.088
08-05-66	1.025	0.13	
08-11-66			0.045
08-23-66			0.040
09-06-66	1.018	0.10	
10-03-66	1.001	0.05	
10-18-66			0.076
11-01-66	1.016	0.14	
12-06-66	1.014	0.17	
02-03-67	1.020	0.08	
03-03-67	1.024	0.12	
05-05-67	1.021	0.09	
06-09-67	1.018	0.08	
07-07-67	1.021	0.15	
08-04-67	1.025	0.05	
09-08-67	1.015	0.12	
10-06-67	1.008	0.05	
11-10-67	1.011	0.11	
12-07-67	1.010	0.12	
01-09-68	1.003	0.01	
03-26-68			0.055
04-05-68	1.005	0.06	
04-16-68			0.046
05-03-68	1.016	0.11	
09-13-68	1.012	0.06	