

WORKMEN'S COMPENSATION
THE INDUSTRIAL COMMISSION OF OHIO

Claim No.

Claimant

SPECIALIST'S REPORT

Address 1776 E. 93rd Street - Cleveland, Ohio

Date of Examination August 15, 1939

Age 52

(DO NOT WRITE BELOW THIS LINE)

NOTE: Submit report under following headings: (1) History. (2) Complaints. (3) Examination, including X-ray and laboratory work. (4) Discussion. (5) Opinion. Mail THREE copies to the MEDICAL SECTION—Retain one for your file.

HISTORY

C.C. "Lead Poisoning"

P.I. On August 8, 1935 while working at Ferro Enamel Company was seized with attacks of vomiting and severe colicky diarrhea. Occasional attacks of abdominal pain in intervals between diarrhea though most of pain was griping (peristaltic) in character and associated with diarrhea. Bowels moved 5-6 times daily and vomiting was frequent over 3 weeks period. On September 30, 1935 weight had fallen from 148 to 112. Seen by physician and diagnosis of plumbism made. Treatment was continued from August 8 till November 1, 1935 and was discharged to return to work. On November 11, 1935 began work but was unable to continue after first day. Weight remained about 112 to 120 lbs. Laid off work and has been unable to do even light work since. Shortness of breath which began in September 1935 has continued since. Most noticeable on arising in A.M. - lasts 2-3 hours - can walk about but feels weak and dyspnoea prevents exertion. Attacks of dyspnoea may come on at any time of day - not related to effort or anxiety - may come on while sitting or eating - occasionally while walking up stairs. No palpitation, precordial or chest pain with attacks.

Remained at home until August 1936 when resumed treatment by physician - continued till October 1936 - remained at home without medical supervision till August 1938 when was hospitalized for 6 day period under observation. States was given medication and low Ca diet. Discharged - remained at home till Feb. 6, 1939 when was hospitalized again for 2 weeks period of observation - discharged again and has remained at home since.

In all of time from Nov. 1935 till present has had alternating diarrhea and constipation, weakness with attacks of mild paroxysmal dyspnoea, weight has remained between 112 and 120 lbs. since. In spring 1939 weight was 130 lbs. for short period - has never returned to original weight. No other symptoms or signs noted.

Occupational

School till 8 - farm till 10 - bakery worker till 12, machinist apprentice till 17, machinist till 22 - auto mechanic till 1933 - Then enamel worker for 2 years till illness in 1935.

Lead Exposure

Worked on driers - sorting and cleaning enamel powder - then bagging. Was very dusty operation - wore respirator which was not efficient - did not taste dust but grit would accumulate in mouth. (Does not know if fellow workmen had plumbism as labor turnover was very high). Pb exposure was minimal while auto mechanic - no soldering or battery repair.

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Family History - Father died 33, accidental. Mother 75 living and well. One
sibling living and well - 3 siblings died - 2 in childhood 1 accidental.
Wife living and well - no children.

Prev. Medical History - Childhood diseases - influenza 1920, no other known.
General health good till P.I.

G.R. Negative till P.I. - Paroxysma dyspnoea since - does not come on when
reclining and is relieved by lying - no hemoptysis, cough or chest pain.

G.I. Appetite fair to poor - small breakfast - Bowels irregular - attacks of
diarrhoea about twice per month lasting 2 days with 4-5 movements daily -
then regular for week or two when will become constipated - Hemorrhoids
for past 30 years -

Robert H. Kelen
Examiner)

M. D.

GU Negative

Diet Eats well balanced diet except for meat which is taken at most 1 x day.
Drinks at least 1 qt. milk daily - Little coffee - 3-4 x week.

Habits - Averages 8 hours sleep - Occasional insomnia and restless sleep - Has had no alcohol past year. Never used alcohol excessively - moderate smoker.

Weight - Usual weight: 148 Best Weight: 152 - 1919 Lowest weight 112 - 1935.

Physical Examination

G.A. Alert, cooperative - does not appear either weak or ill - fairly well nourished, muscular development and tone good - is active and well coordinated - Color slightly pale - facies and dilated nasal venules suggest alcoholic indulgence - no dyspnoea.

Temp: 98.8 Pulse: 73 R: 24 Weight 123 lbs. Grip Rt: 70
Lft: 60

Blood Pressure Rt: 105/60
Lft: 100/62

Head: Hair sparse - anterior baldness - tortuous and sclerotic temporal veins.
Ears: Drums clear - reflex good - canals normal - several lines of injection superior anterior quadrant left drum.

Eyes: Pupils myotic - react well to light and accommodation; margins irreg.
Early cataract opacity lenses - E.O.M. ok.

Nose: Markedly dilated venules - m.m. reddened - dry - no discharge.

Mouth: General gingivitis - 2 badly carious teeth - others with small cavities - poor occlusion - several marginal deposits which are seemingly related to the gingivitis - the deposit at the lower left first molar is punctate, pale bluish and resembles a lead deposit. Tonsils small - scarred soft palate distorted by scar - anterior pillar reddened - pharynx injected.

Neck: negative

Chest: R.S.D. 7.0 R.C.D. 3.5 x 10.0 Apex rate: 90 25 hops 110 - 2 min. 90

Chest clear - heart tones clear - regular - reaction good.

Abdomen: muscular - no masses or tender areas -

Pelvis: negative

Extremities: negative

Reflexes: Superficial reflexes not elicited - tendon reflexes hyperactive (+++) - no clonus - no ataxia.

Musculature: fairly well developed - symmetrical - no tremor - muscular strength fairly good.

Mental: talkative - cooperative - given to reminiscences and wordy detail - some confabulations appear in statements of daily activities.

Laboratory

Urine: sugar - negative - Alb. negative Diacetic - negative
Indican - negative
Acetone - negative

Pale, clear, Sp.Gr. 1.001

Microscopical: negative.

Lead Concentration: (small sample 8-15-1939) 0.64 mgs./liter
(Large " 8-28-1939) 0.49 mgs./liter

Blood: WBC 8,350 RBC 4,010,000 Poly. 50.5 Band. 2.0 Lymph 40.0
Hb 84% Stippling: 9 per 50 fields Baso. 1.0 Eosin. 2.5 Mono. 4.0

Lead concentration:

0.065

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Lead concentration:

1121032

2 samples

{ 0.065
0.06 mgs./100 grams 8-15-1939

(9 stippled cells per 50 fields is equivalent approximately to 720 per million erythrocytes, by our method. It is not remarkably high, since such results are seen occasionally in normal persons.)

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atory (continued)

Feces: Lead Content of Sample 0.14 mg.
Lead Concentration 0.20 mg/gram of ash

Discussion and Conclusion

The quantities of lead found in the blood and urine are wholly inconsistent with the occupational history and more particularly with the fact that there has been no occupational lead exposure since August of 1935. Such levels of lead concentration are found only in immediate time relationship to severe lead exposure, and in our experience are never seen for longer than a few weeks after the cessation of such exposure. Indeed the urinary lead concentration as determined twice was found to be unusually high even under conditions of very severe lead exposure. In our opinion the results of these determinations bear no relationship to the occupational lead exposure of two years before, but have arisen from some different and undetermined source of lead absorption which took place during the past few weeks or days.

The deposit on the gum tissue, a secondary anemia, and a relative lymphocytosis are compatible with the occurrence of some recent lead exposure, while the absence of large numbers of stippled erythrocytes in the one smear examined is not necessarily significant, there being no definite relationship between numbers of stippled erythrocytes and the degree of lead absorption in the individual case.

There is no adequate evidence of clinical illness or disability which is in keeping with the high analytical results. In fact there is little or no evidence of illness or disability, or of chronic disease, except that certain stigmata of chronic alcoholism are visible. In our opinion there is no disability of occupational origin.

Recommendations.

The source of the lead exposure which has been demonstrated by the analyses is purely conjectural, and since such results were not anticipated from the clinical findings, no further study was carried out to determine the source. It is believed that a brief hospitalization of this man, with suitable studies of his lead metabolism (under controlled conditions of lead intake) would answer the question now presented. It is strongly recommended that such steps be taken to determine whether or not this is an exceptional case of lead retention (we are confident that it is not) or whether some inadvertent or perhaps intentional lead absorption has occurred.

Robert A. Zahoe

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