# Lead Poisoning From Snuff*†

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NOTWITHSTANDING education, publicity, and restrictive measures lead poisoning has become the chief industrial hazard. Since compounds of lead frequently contaminate drinking water, beverages, cosmetics and a variety of other substances used by mankind, it has become equally as important a factor in causing distress and sickness among the non-industrial population. New sources of lead poisoning are constantly being uncovered so that it behooves the Medical Profession to be forever on the watch for this important disease and its manifestations. Within the past year another source of lead poisoning, while by no means new, should be newly emphasized in this country.

Model[1] in 1784 first drew attention to the possibility of lead poisoning from snuff-tobacco. In 1843 Otto[2] of Copenhagen reported two cases, one of which was fatal. The victim was a botanist and scholar, who suffered from obstinate constipation, abdominal cramps, headaches and who finally became comatose and died. Not until after his death was lead poisoning suspected and then an analysis of the brand of snuff he was in the habit of using, showed considerable lead. For some years following this a number of articles appeared in the foreign literature dealing with this source of lead poisoning, for in 1886 Billings[3] collected a total of 23 references, including 5 cases reported by Mayer[4] and 19 cases by Sonnenkalb.[5] About this time Garrod[6] in a clinical lecture on "Lead Poisoning" emphasized the possibility of lead poisoning occurring in warm climates when moist snuff is packed in lead covered boxes. He reported a case of an Englishman who had just returned to London from India on account of an illness, the cause of which was traced to lead found in snuff. In 1904 McCaw[7] added six more references, which included an interesting case of aphonia caused by lead poisoning from snuff reported by Ormsby[8] of New York. In 1912 Stadler[9] of Switzerland reported that a certain metal-foil wrapper contained 89.0% of lead and the moist snuff contained 1.75-1.90% of lead. Habitual use of this brand of snuff by a woman caused fatal intoxication. In 1918 four cases were reported in America, three cases by Uttal[10] of New York City and one case by Bauer and Ropes[11] of Boston. All in all about forty cases have been reported.

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There are three possible causes for lead in snuff-tobacco: (1) adulteration in manufacture, (2) lead wrappers, and (3) lead compounds used as insecticides.

The manufacture of snuff[12] consists in moistening tobacco leaves and stalks with salt water, and leaving them to ferment in an open chamber for some months. Then the tobacco is ground to a powder, moistened again and put into a closed wooden chamber to undergo a second fermentation process. This destroys about two-thirds of the nicotine, and the malic and citric acids in the tobacco, while the acid and bases evolved leaves free ammonia in the snuff. Various flavors are added to give scent. Quicklime is often used to give a biting, desiccating effect. Formerly lead oxide and chromate were added to give a lighter color and greater bulk, and thereby increase the sale value. Snuff is packed and marketed either in a dry or moist state, depending upon the amount of moisture added during the fermentation. In 1849 Hassel[13] examined 43 kinds of snuff; nine brands contained lead chromate in amounts varying from 1-4.5%; three specimens contained lead oxide. Since tobacco products do not come within the Federal Food and Drug Acts there is no adequate legislation to prevent adulteration at the present time.

Another source of lead in snuff and the one most referred to in the literature is due to the usage of lead-foil for wrapping and lead-tin boxes for packing. Stadler[9] (already referred to) reported 89% of lead in a metal-foil wrapper used for snuff even though the Swiss law prohibits a content greater than 1%. Wicke[14] found that the outer crust of snuff packed in lead-foil contained up to 2.7% lead even if the foil was lined with tin on one side. When the snuff is damp or contains acetic acid, absorption of the lead from the wrapper is apparently increased.

The third possible source of lead in snuff is due to the fact that lead-arsenate is used rather extensively as an insecticide in the growing of the chief types of tobacco used in the manufacture of snuff.[15] 1927 Remington[16] examined a large number of brands of American smoking and chewing tobacco and found arsenic to be invariable present in quantities many times greater than the amounts cited as being permitted in foods. It is possible that the source of the arsenic was due to the arsenate used in the plant spray and that lead may also find its way into tobacco for the same reason.

During the past year and a half I have seen four cases of lead poisoning from snuff. All were middle aged white women admitted to the charity ward of John Sealy Hospital. Analyses of the snuff, feces and urine of these patients showed lead. All chemical analyses were made by Professor B. M. Hendrix of the Department of Biological Chemistry. The patients all used the same brand of snuff which we believe was adulterated with lead chromate. The samples examined were marketed in glass jars.

The first case will be considered last. Case No. 2 has been free from symptoms now for about a year since she stopped taking snuff. Case No. 3, shortly after entering the

hospital died from an inoperable carcinoma of the cervix, hydronephrosis and uremia. She gave a history of taking snuff for 20 years. Twelve years previous to entering the hospital she had been advised to take radium treatment for her pelvic condition. What the diagnosis was at that time could not be determined. Post mortem examination confirmed the clinical diagnosis and a short while after the organs had been preserved in formalin it occurred to us to test certain of the soft tissues for comparative lead content. Analysis of the liver tissue showed 3.31 mgs. of lead per 100 gms. of tissue; heart muscle showed 4.42 mgs. of lead per 100 gms. of tissue, the carcinoma contained 7.34 mgs. of lead per 100 gms. of tissue. Not enough of the cancer was available for a duplicate determination, but we feel that the proportion of lead found in these tissues is reasonably correct. This suggests a greater affinity for lead on the part of carcinomatous tissue as compared to other soft tissues, but requires further investigation. Case No. 4 is in the hospital at present. She came in complaining of weakness, abdominal cramps and pains in her limbs. The snuff this patient was taking showed on repeated analysis 0.35% of lead chromate, or 0,224% lead.

### Report of Case No. 1

November 4, 1927, Mrs. L. K. an Irish-American housewife, aged 44, came to the John Sealy Hospital complaining of weakness and vomiting.

Following an attack of "influenza" six months previous there had been increasing soreness and progressive weakness in her lower limbs. For two months there had been attacks of severe epigastric pains which were referred to the back. With the pain was nausea and vomiting irrespective of meals, until finally she was unable to keep anything on her stomach. Besides this there had been dizziness, increased thirst, constipation, oliguria, and edema of the feet. For some time her sense of taste had been impaired. At the beginning of her illness her weight was 250 pounds; at time of admission it was 180 pounds.

She had measles, mumps and typhoid fever when a child and at one time she swallowed lye, which resulted in an esophageal stricture, for which numerous dilatations were performed. At the age of 18 an "abscessed ovary" was removed. All her life she had drunk a fair amount of beer and for the past 2 years considerable whiskey. She had never been pregnant.

Physical examination showed a middle-aged white woman, apprehensive with a pained expression and pasty appearance. Teeth were dirty and the gums showed advanced pyorrhea. Tongue was coated, and the sense of taste for sweet and sour was impaired over the anterior two-thirds.

Cardio-respiratory system was normal except for hardening of the arteries and a soft blowing systolic murmur localized at mitral area. Systolic blood pressure was 120, diastolic 86. There was generalized muscular weakness. The lower limbs were flabby and extremely tender to touch over calves and thighs. Knee jerks absent. All the other reflexes were normal. Pelvic examination showed atrophy of vagina and uterus.

The urine was acid and showed a low specific gravity and an occasional hyaline cast. No albumin or sugar present. A satisfactory gastric specimen could not be secured. The vomitus showed bile stained mucous and little else. Red cell count was 4,350,000; Hgb. 75%. White cell count 7,900. P 71; L 27; Tr. 2. Wassermann negative. Basal metabolism normal. X-ray of esophagus and stomach showed no esophageal stricture, but there was delayed gastric drainage, and a dilated duodenal bulb with a tortuous and angulated second part, suggesting adhesions. Intravenous injection of tetra-iodophenol-thalein salts fol-

lowed by films revealed no gall bladder shadow.

On November 2nd, a cholecystectomy and appendectomy was done. The gall bladder was freed from adhesions reaching to the duodenum. Subsequently a pathological diagnosis of chronic atrophic cholecystitis was rendered.

Following the operation there was slight improvement and when the wound was healed she was allowed to go home.

On December 14th the patient was readmitted in a very weak and dehydrated condition. She had been unable to keep solids or liquids on her stomach since leaving the hospital. She now complained of sharp shooting pains in her hands and legs, with numbness and sensation of pins and needles in the fingers and toes. She appeared very much dehydrated; the skin was dry; eyes were sunken and the tongue was bright red in color. There was a marked atrophy of the interrossei muscles of the hands with incoordination of movement. The lower limbs were held in a flexed position and complete extension was impossible because of pain and spasticity. There was extreme muscular tenderness over the lower extremities. The slightest pressure on the toes caused excruciating pain. There were no signs of local inflammation.

Further laboratory work showed the phenolsulphonephthalein elimination by the kidneys to be 55% in 2 hours. A Mosenthal test showed the maximum specific gravity of the urine to be 1.006, with a variation of 5 points; the 24 hour amount was 2,760 cc.; the day to night ratio was 1:1. The red cell count was 3,720,000 and the hemoglobin was 60%. The white and differential count was normal. Basophilic stippling was present on an average of one cell to every two oil-immersion fields. The T. N. P. N. of the blood was 38 mg.; Uric acid 8.4 mgs.; creatinin 1.5 mgs.; chlorides 530 mgs.; Serum calcium 9 mgs.; per 100 cc. of blood. Spinal puncture showed a normal fluid pressure. There were 7 lymphocytes to the cu. mm. Globulin was markedly increased and the Wassermann was negative. Stool examinations were negative. X-ray examinations of the stomach, knees, spine, and pelvis were negative. At this time it was learned that the patient was, and had been addicted to the use of snuff for the past 35 years, and for the last 8 months owing to fits of mental depression she had been in the habit of keeping snuff under her lip both day and night. The saliva was not expectorated. Twice a week she purchased an 8 oz. jar of snuff. Recently she had taken considerable more whiskey and beer than ever before. Analysis of the snuff showed approximately 0.2% of lead. Lead was also found in the urine and feces of the patient.

The snuff was taken away from her and with rectal feedings and the administration of sedatives the patient improved and soon was able to take a bland diet. During the next few weeks she showed considerable improvement. Then an attempt was made to put her on a low calcium diet with the administration of ammonium chloride. This was discontinued in a short time because of the poor appetite and the mental dissatisfaction. The patient remained in the hospital until the latter part of February showing some improvement in nutrition on a milk diet and symptomatic treatment. Against our advice she left the hospital.

On April 10th the patient was again admitted to the hospital and showed all the manifestation of a long wasting illness. Her husband stated that in spite of all precaution the patient had gotten hold of snuff and had been taking it as before and with the taking of snuff, her appetite failed and nausea and vomiting had again set in. There were no significant changes in the physical signs except those related to the mental and nervous systems, with the exception of a moderately severe cystitis. The neurological examination made at that time follows in full:

The patient lies partially on back and side with head moderately flexed on thorax, back partially arched, with legs drawn up. The attitude and expression are constantly changing. For a moment the facies are anxious and staring and the patient is restless, then she becomes more quiet and the facies apathetic and vacant. When the patient cries the upper lip is drawn peculiarly

toward the nose giving a hysterical appearance. The mood of the patient seems to be changing constantly. It is difficult to hold the patient's attention for even a very short time. Intelligence and orientation fair. Memory for recent events is poor. Periods of drowsiness alternate with periods of restlessness. The patient dreams much, particularly pertaining to her family relationship; she states that she has visions while she is "half-awake" of angels coming after her; she says her mother (long dead) talks to her almost constantly. The speech is drawling, articulation is fair.

All the cranial nerves are normal except for diminished sense of smell and taste. Fundi normal.

Motor System: Some atrophy and moderate flaccidity of all muscles of forearm and carpae interossei. Motor power is markedly decreased, particularly on right arm and hand; there is no evidence of paralysis in the upper extremities, the change is principally a weakness or loss of power, or strength. There is present a fair amount of ataxia in the arms; lower extremities are too weak and painful to permit performance of tests, though there is apparently a lower motor neuron paralysis of both lower extremities with contractures of the muscles in flexion.

Reflexes: The deep tendon reflexes of jaw, elbow and forearm are present and slightly diminished. Those of lower extremities can not be elicited because of pain.

The superficial of corneal and pharyngeal reflexes are normal. Those of abdomen not present. There is vesical and rectal incontinence. The skin suggests atrophic changes. There is pitting edema of the feet.

Sensory System: Hyperesthesia over soles of feet. Diminished sensation of pain and touch in both forearms. Disturbed sensation of pain and touch in both legs to knees. Hyperesthesia of palms. Disturbed appreciation of hot and cold below 5th rib. (This is inconstant.)

The patient continued to grow constantly worse and finally developed hypostatic pneumonia and died, July 4th, 1928.

The Clinical Diagnosis was:

Plumbism.

Posterior Root Radiculitis.
Myositis.
Hypostatic Pneumonia.
Aortitis.
Chronic Interstitial Nephritis.
Cystitis.
Atrophy of the organs of reproduction.

### Autopsy Report

Drs. P. Brindley and C. B. Sanders.

The body is that of an elderly white woman about fifty years of age. The body is embalmed and rigor mortis is present. The pupils are dilated and equal. There is sordes about the mouth and the gums show a bluish stain along their margins about the teeth.

There is marked emaciation of the entire body especially of the legs and arms. There are contractures of the legs and arms and they cannot be straightened out. There is rather marked edema of the feet and legs so that they pit on pressure. There are bed sores over the buttocks. On the anterior abdominal wall to the right of the mid-line and extending up from the umbilicus there is a scar about 15 cm. long. Dense fibrous adhesions are present between the under surface of the liver and the transverse colon. There is absence of the gall bladder and appendix. Rather dense fibrous adhesions between the capsule of the liver and the surrounding structures.

*Heart*: Gross: Weight 200 grams. The right side shows a slight amount of dilatation. All of the chambers are filled with post mortem clots. The mitral and aortic valves show a slight thickening of their margins. There are several raised yellowish patches in the first part of the aorta. Microscopic: some of the muscle fibers appear

larger than normal and show large nuclei, but many of the fibers are of normal size or smaller than normal. There is apparently a slight increase over the normal of the golden yellow pigment found at the poles of the nuclei. A slight overgrowth of connective tissue is present.

*Lungs*: There are a few petechial and ecchymotic hemorrhages beneath the pleura of the left lung. The posterior portion is of a dark red color and on cut section shows an excess of blood tinged fluid. There are several areas in the lung which show a decrease in crepitus. The right lung is similar to the left and in addition shows, on cut section, multiple areas of consolidation which stand out above the neighboring cut surface. There are several small greyish nodules in the upper lobe which look like healed tubercles. There is a small dark red wedged-shaped area in the lower margin of the upper lobe. The mediastinal nodes show greyish areas of caseation and also a large amount of coal pigment. Microscopic: the vessels are filled with blood. Some of the bronchi contain many polymorphonuclears and desquamated epithelium. Neighboring alveoli also contain a similar inflammatory exudate while many of the alveoli still further away from the bronchi are partly or completely filled with a serous fluid intermixed with fibrin and red blood cells. A few pigmented cardiac failure cells are present. The mediastinal nodes contain black granules of pigment, apparently coal pigment. There is also a definitely walled off partially healed conglomerate tubercle.

*Spleen*: Gross: weight 338 grams. On section there is an excess of blood and fibrous tissue. The pulp is soft and can easily be scraped away by the knife. Microscopic: there is a rather marked excess of blood. The pulp is increased in amount and there is a marked amount of yellowish brown pigment both within the phagocytic cells and between them. The central splenic arteries are thickened and fibrosed with hyalinization of the fibrous tissue. There is fibrous tissue over-growth with hyalinization in the trabeculae.

*Liver*: Gross: weight 1,700 grams. The liver has rounded edges. On cut section there is an excess of blood and the liver has a mottled nutmeg appearance. Microscopic: in scattered areas the liver cells show a fatty infiltration.

*Kidneys*: Gross: weight 200 grams. The capsule strips with difficulty leaving a roughly and finely granular surface with several subcortical cysts present which are about 10 mm. in diameter. On cut section there is a slight thinning of the cortex. The right kidney is similar to the left. Weight 200 grams. Microscopic: the vessels are distended with blood. Some of the glomeruli show enlargement. Congestion of the glomerular vessels, and swelling of the endothelial cells. The tubules, especially the loops of Heule and collecting tubules, show degeneration, desquamation and some necrosis. Some increase in interstitial tissue is seen especially in the cortex. Adrenals: show nothing unusual.

*Pancreas*: Gross: shows nothing unusual. Microscopic: there is an increase in fibrous tissue in the walls

of the ducts. The vessels are distended with blood.

*Genito-Urinary System*: Gross: the bladder contains about 50 cc. of a turbid urine. The mucosa is congested and shows many petechial and ecchymotic hemorrhages. There is a deposit of a muco-purulent exudate on the mucous membrane.

*The Uterus*: Gross: is smaller than normal and the walls are atrophic and fibrosed. The ovaries are small and fibrotic. Microscopic: the uterine muscle is atrophied and there is an increase in fibrous connective tissue. The glands are atrophic. There are many lymphocytes in the endometrium and a less number in the myometrium.

*Gastro-Intestinal System*: Gross: the stomach contains a small amount of mucus and food. There are many petechial and ecchymotic hemorrhages in the mucosa. The rectum contains dark black fecal material. There are some petechial and ecchymotic hemorrhages in the mucosa of the colon and small intestine. An excessive amount of mucus is present in the color. There are also a few small ulcers in the colon. Microscopic: small hemorrhages are seen in the mucosa, and in areas we find absence of superficial portions of mucous membrane.

*Central Nervous System*:* Gross: the brain and cord show nothing unusual grossly. Microscopic: different sections of central nervous tissue, posterior root ganglia and periphereal nerves were stained with Harris' hematorylin and phloxine; Heidenhain's iron hematoxylin; and Weigert's myelin sheath stain.

The brain showed nothing unusual.

The spinal cord showed many of the anterior horn cells atrophied and degenerated with a faint yellowish rather diffuse pigmentation that in some instances completely replaces the faded nucleus. Certain ganglion cells in the nucleus dorsalis showed slight evidence of degeneration. The posterior root ganglion cells contained a large amount of yellowish brown definitely granular pigment arranged perinuclearly. The ganglion cells otherwise stained well.

The periphereal nerves showed nothing unusual.

*Anatomical Diagnosis*: Atheroma of the aorta; Chronic mitral and aortic valvulitis; Chronic fibrous pleurisy; Hypostatic congestion and pneumonia; Hypostatic congestion; Tuberculous lymphadenitis; Chronic interstitial splenitis; Chronic glomerulo-nephritis; Atrophy of the uterus, tubes and ovaries; Petechial and ecchymotic hemorrhages of mucous and serous membranes; Hemorrhagic cystitis; Surgical absence of the gall bladder and appendix.

*Microscopic Diagnosis*:

*Heart*: slight hypertrophy, with beginning later atrophy.

*Lungs*: hypostatic, lobular pneumonia, tuberculosis of hilus nodes. Anthracotic pigmentation of nodes.

*Spleen*: congestion, increased pigment, with a slight chronic interstitial splenitis.

*Liver*: fatty infiltration, slight.

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