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Saturnism, Pediatric and Adult Lead Poisoning

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INTRODUCTION

Saturnism or plumbism with all its deleterious effects on the human body was well known to the Greeks and Romans, and observations by modern man have only extended our knowledge of the processes involved in the intoxication. Industrialization has increased the avenues by which humans can be exposed to lead, particularly those involving the environment. On the other hand, modern industrial hygiene has also kept pace to reduce lead exposure, and the development of specific antidotes and better diagnostic tests have decreased the number of fatalities. One of the main difficulties in the rational discussion of plumbism is the fact that lead is a toxic element and people tend to equate even minute lead exposure to active plumbism. This is not true because the level of exposure determines the degree of intoxication, and each individual has body defenses that tend to maintain the body lead balance at very low levels throughout the individual's lifetime unless massive acute or high chronic exposure occurs. It is our purpose to discuss the biochemical and physiological aspects of both pediatric and adult plumbism and try to point out the accepted methods of diagnosis and treatment for lead intoxication.

PEDIATRIC PLUMBISM

The manifestations of pediatric plumbism differ considerably from those of the adult disease, and its sequelae and mortality rate are fairly high.

In Philadelphia over a 14-year period, there were 804 cases of pediatric plumbism with 76 deaths and 82% of the cases were in the 12 to 36 months of age group [10]. In general, pica is usually the common denominator in pediatric plumbism [9,11,12], but lead fumes also make a contribution [8,13].

Signs and Symptoms

Pediatric plumbism manifests itself in three symptom complexes; hematological, gastrointestinal, and neurological. The individual patient may have one or all of them.

From the hematological viewpoint, the usual findings are anemia with hemoglobin of less than 10.5 gm coupled with microcytosis and hypochromia. There may also be stippled erythrocytes, but not in every case. Moreover, a failure to respond to iron therapy adds further evidence to the possibility of plumbism.

The gastrointestinal symptoms of recurrent emesis and vague abdominal pain and constipation, which simulate nonspecific gastroenteritis, are difficult to connect with plumbism. Both the gum lead line and black stools are rarely seen.

The most serious manifestations of pediatric plumbism are those involving the central nervous system. They vary from drowsiness to coma to grand mal seizures. Children showing clumsiness, repetitive loss of balance, ataxia, or convulsive seizures should be examined for plumbism. These seizures are variable, being one-sided, alternating sides, or generalized. Physical examination may reveal papilledema, ataxia, lethargy, or seizures with or without reflex changes or local paralyses. In 23 recently reported cases of lead encephalopathy, death occurred within 72 hr of the onset of clinically apparent encephalopathy [14]. It has been suggested that the underlying cause of this encephalopathy is lead's well-known interference with porphyrin synthesis resulting in chronic metabolic hypoxidosis and in addition impedence of brain hemodynamics. Tissue changes consist of dilated capillaries with swollen endothelial nuclei protruding into the lumen; microglial nodules spreading out indiscriminately over various gray areas and also over the white matter; loose accumulations of microglia cells were often seen in the molecular layer of the cerebellar cortex. Nerve cell bodies were involved, and there were changes in the white matter, the

blood vessels, and leptomeninges. Inflammatory changes range from perivascular cuffing to granuloma formation, but it is the molecular layer of the cerebellar cortex that shows the greatest damage in lead encephalopathy [15]. An electron microscopic study of biopsy specimens from six children with lead encephalopathy indicated changes in the neurons in the gray matter and in the structure of the capillary endothelium. In the white matter, changes in the capillary endothelium reduced the extracellular spaces. Glial cell nuclei exhibited an exhaustion phenomenon. Changes in the myelin sheath occurred at the intraperiodic line with a breakdown of the myelin lamella and accumulation of electron-dense granules. Axonal changes consisted of a loss of neurofilaments and their replacement by diffuse granular-appearing material [16]. These changes can in part be related to respiratory arrest and increased intracranial pressure. Such alteration in physiological functions can lead to serious neurological deficits, but not in all cases, as has been shown by a Chicago study where complete recovery occurred in 257 of 405 children. The remaining 168 children had neurological sequelae distributed as follows: 93 mental retardation, some with pre-existing mental retardation; 85 recurrent seizures including grand mal, Jacksonian, myoclonic, akinetic, and vegetative types; 9 cerebral palsy, with spasticity in 6 and extrapyramidal forms in 3; 5 optic astrophy, with retrobular neuritis in 1, corneal opacities in 1, bilaterial ptosis in 1, and strabismus in 3. All of these cases were produced by pica or inhalation of fumes from burning battery cases, and there was no significant difference in response between the routes of exposure [17]. One of the difficulties encountered in the assessment of residual effects is obtaining an estimate of the duration of lead exposure and the possibility of recurrent exposures. It has been shown that 19% of the 229 cases examined in Cincinnati over a five-year period were recurrent episodes. Blood lead in most cases was at the upper level of normal, but it must be remembered that it is easier to maintain a toxic level than to achieve it [9]. The residuals of pediatric plumbism include behavioral problems, poor discipline, inability to comprehend, lower IQ, and late chronic nephritis [6,9,18-21]. This latter condition has been found in Queensland and Serbia, but not in the United States, and thus the situation remains open to further research and discussion [22].

Diagnosis

Diagnosis of pediatric plumbism is difficult because the symptoms are so nonspecific; the following criteria have proved useful: presence of the source of the poison (pica or fumes); clinical observations; indications

of lead absorption and laboratory findings [23]. In New York City, where lead poisoning caused death in 367 cases and mental and neurological disturbances in 459 cases over an eight-year period, the following diagnostic procedures have been instituted. Identification of pica history with a blood lead of 60 μg/100 ml but no overt symptoms indicates possible lead poisoning. Definite lead poisoning is indicated if in addition to the above, two or more of the following symptoms are observed: gastrointestinal disturbances, anorexia, vomiting, constipation, pallor, anemia, irritability, seizures, or increased long bone density or abdominal opacities on roentgenologic examination. Urinary coproporphyrin analyses are also performed [24]. It has been suggested that a calcium disodium edetate mobilization test also be done to assist in revealing an increased body lead burden [25]. In a study on 17 children with subacute lead poisoning, the EDTA mobilization test causes an excretion of 2400 μg in the first 24 hr in 16 of them [26]. Another test suggested for diagnosis of early lead exposure in children is the determination of urinary aminolevulinic acid levels. Normal values range from 0 to 0.66 mg/100 ml. It was also pointed out that whereas this test correlated with lead poisoning, the coproporphyrin test gave false positive results in one out of four cases [2]. From the moment of actual diagnosis of pediatric plumbism, it is mandatory that the patient be removed from the contaminated environment and steps be taken to prevent recurrence of the toxic episode [24,27]. It is highly significant that to this date, no case of pediatric plumbism has been definitely connected with exhaust effluent from automobiles burning lead gasoline.

Therapy

Therapy for pediatric lead intoxication is much more definitive now than it has been in the past. Edetate 12.5 mg/kg/dose intramuscularly with procaine every 4 hr for 5 to 6 days coupled with dimercaprol 4 mg/kg/dose intramuscularly for the same time period. The child must have a good urine output. Edetate therapy in the first 24 hr may cause an exacerbation of an encephalopathy. Children with a blood lead above 0.1 mg/100 gm or with a persistent coproporphyrinuria following the first course of edetate should receive one or more additional courses at 5-day intervals. Cerebral edema may be reduced with 1 to 1.5 gm of urea, as a 30% solution, given at 8- to 12-hr intervals [9]. The results obtained in the urea treatment have been somewhat equivocal [28], but not nearly as controversial as a radical craniectomy. In Chicago, where the death rate from lead encephalopathy was 25% during the period 1959-1963, the use of such a procedure increased the death rate to 45.4% in 1960 [29]. These

Investigators now utilize urea in doses of 1 gm/kg every 8 hr for about 3 to 4 days. They also used edetate in doses of 60 to 75 mg/kg intravenously or intramuscularly or substituted penicillamine for the edetate [29]. In another study, they showed that paraldehyde, chloral hydrate, or chlorpromazine could control the seizures in lead encephalopathy, but barbiturates were contraindicated. Moreover, hypothermia, coupled with a corticosteroid for 3 to 4 days, was helpful in reducing cerebral edema and brain volume [30]. Others have used chelation therapy combined with dexamethasone and have substituted 2.5 gm/kg of mannitol for urea in the successful treatment of pediatric lead encephalopathy [31]. It has also been suggested that because an abnormal body burden of lead may persist for months or years after the acute episode and excessive lead intake has been halted, that periodic treatment with chelation agents could be used to return the soft tissue lead concentration to a more normal level remembering that such therapy can initiate an acute attack of plumbism [25].

ADULT PLUMBISM

Plumbism in the adult presents a different problem here from that in the child; in most cases it is the result of poor industrial hygienic practices or of handling lead at home or in a small workshop under less-than-ideal conditions. Poisoning has also occurred in the use of inorganic and organic lead salts as stabilizers in the plastics industry [32]. Both ceramic glaze and enamel jewelry can cause plumbism and in the latter case results from mouth-pointing the implement used for lifting the enamel [33,34].

Signs and Symptoms

It should be borne in mind that adult or chronic plumbism requires months to years to develop and depends upon both increased exposure to and absorption of lead until the critical amount is reached that will induce active plumbism. During this incubation period, the individual will be below par with nonspecific symptoms which do not actually prevent his working. The symptoms of chronic plumbism include headache, generalized muscle pains, constipation, abdominal tenderness and pain, colic, emesis, infrequently diarrhea, anoxeria, weight loss, bad taste in the mouth, and fatigue [35]. Other symptoms referable to the hematopoietic system include mild to moderate microcytic hypochromic anemia, hemoglobin varying from 8.1 to 12.8 gm, hematocrit varying from 28.8 to 43.6, erythrocytes 3.45 to 5.36 million/mm^3, mean corpuscular volume

70 to 90 μ, mean corpuscular hemoglobin concentration 27 to 36 gm/100 ml, reticulocytes 1.5 to 11.6%, stippled erythrocytes 0.1 to 7.5% and an icterus index of 4 to 10. There is also a change in the osmotic and mechanical fragility of the erythrocytes [36]. The urine will contain an increased coproporphyrin and possibly some porphobilinogen [37]. There is also the possibility of jaundice [38]. Nephropathy has long been associated with lead poisoning in Europe and Australia but has not been definitely shown in the United States except in some cases linked to the drinking of illegal whiskey [39,40]. It has been pointed out that although lead has been implicated in the development of proximal tubular damage leading to chronic nephritis, more definitive information is necessary to indisputably identify lead as a cause of chronic nephritis in subclinical plumbism [41]. At present one can only conclude that, in the United States, evidence for lead nephropathy is highly circumstantial compared to other types of kidney lesions caused by drugs and chemicals [42]. This lack of renal lesions may be the result of the lack of long-term heavily exposed workers because of better industrial hygienic practices in the United States [43]. Similar results have been reported from England, where previously there had been many reports of renal damage from lead [44]. The situation in the balance of Europe regarding lead nephropathy is the continued reporting of many cases of renal lesions leading to hypertension [5,45-52]. Another sequelae of chronic lead nephropathy is gout, and it has been shown that such patients have a high plasma urate level that can be exaggerated on a high purine diet. It has been suggested that lead-induced changes in the tubules produce a defect in tubular urate secretion leading to hyperuricemia and a predilection to gout [53]. Others have suggested that increased pruine turnover in lead poisoning may be the cause of gout under such circumstances [54].

One of the most controversial signs of lead intoxication, other than basophilic stippling, is the lead line on the gum. Pigmentation of the gingival margins may also be caused by infectious gingivitis, normal pigmentation of the dark-skinned races, bismuth and other metals, and dental discoloration [55]. Moreover, good dental hygiene makes the observation of the lead line much more infrequent than in the past [35]. Polish investigators have also reached the same conclusion [56]. However, the lead line is an accepted aid for diagnosis of plumbism in Spain and Denmark [57,58].

Of greatest importance in chronic plumbism is the neuromuscular involvement so often seen after prolonged and grossly excessive absorption of lead accompanied by muscular activity. The paralysis usually involves the extensor muscles. The extraocular muscles of the eye and

the extensors of the leg and foot may be involved, but the most common sign is wrist drop. Lead palsy usually is unilateral, but occasionally it may be bilateral. Lead peripheral neuritis must be distinguished from that seen in infection, arsenic poisoning, malnutrition, and diabetes. Lead encephalopathy from massive exposure to lead, dust or fumes, must be distinguished from infection of the meninges or brain, parasitic infections, tuberculosis, syphilis, and uremia [55]. Similar findings have been reported from India where a case of bilateral wrist drop was seen [59]. A Russian report has related such effects to the destruction of the radial nerve with less involvement of the median and ulnar nerves, and it was suggested that the selective action of lead was related to the different structures of nerve fibers [60]. However, such changes may be related to selective damage to the arteries supplying the radial nerve [4]. One of the over-all effects of lead on the ulnar nerve is the reduction in conductance velocity, and some such changes have been observed prior to the appearance of wrist drop [61]. Lesions of the optic nerve from exposure to excess inorganic lead have been reported in Germany, India, and Russia [62-64]. In lead encephalopathy with epileptic seizures, the electroencephalogram may or may not show an abnormal pattern even when changed reflexes indicate central nervous system deterioration [63,65]. However, it is possible to have an irreversible myelopathy without any detectable damage to the brain.

Besides local irritation of the stomach and gastrointestinal tract by soluble lead salts, lead can produce stomach spasms, hypersecretion, and volvulus [66]. On the other hand, hyposecretion of both hydrochloric acid and pepsin as well as intrinsic factors have been reported by Polish workers [67]. Thus, the over-all effects on inorganic lead on the stomach are open to debate.

There can be little doubt that chronic exposure to lead of pregnant women is detrimental to the fetus. Besides disturbances in ovulation, amenorrhea, and sterility, it has been shown that women typographers had three times more abortions than those not exposed to lead and many had stillbirths [68]. The rate of premature and stillbirths is a function of higher exposure to and absorption of lead. Similarly the degree of damage to the fetus follows the same course [69]. Excess lead in the water supply of pregnant women has resulted in seven miscarriages out of eight pregnancies in one woman and in another a baby with congenital nystagmus and partial albinism. Limiting the lead intake increased the number of live and healthy births, thus indicating the high degree of susceptibility of the pregnant woman and her fetus to lead [70]. Regardless of this susceptibility, no cases have been reported thus far to indicate that the amount of lead in the ambient air has had any effect on pregnancy.

Diagnosis

Diagnosis of lead intoxication when a known exposure has occurred, or the patient is known to be working in some branch of the lead industry, is comparatively easy [35]. However, this disease has no place for the amateur diagnostician because lead poisoning is mimicked by many other diseases, and in the absence of elevated blood and urinary lead, anemia, and coproporphyrinuria something other than lead is the causative agent for the symptoms observed [71]. Numerous tests are available to assist in the diagnosis of chronic plumbism, and they cover the range of symptoms previously discussed. Both a physical examination and roentgenograms of the gastrointestinal tract will indicate the degree of intestinal spasm associated with lead colic [72]. Radio iron, ^{59}Fe, uptake and exchange can be used to supplement hemoglobin determinations to reveal the degree of increased hemolysis, retardation of hemoglobin synthesis and regeneration of circulating erythrocytes [73]. Erythrocyte survival time can be determined with ^{51}Cr-tagged red cells, and plumbism causes a slight decrease in survival time [74]. Further evidence of plumbism is the presence of stippled erythrocytes [75]; however, it is not always possible to establish the level of stippled cells in the individual, and the method used in preparation of the blood slide may artificially induce formation of stippled cells [76,77]. The method of staining and dye concentration can cause errors, because under some circumstances only reticulocytes and not basophilic erythrocytes are stained by methylene blue [78]. The variations in stippled cells from one area of the slide to another also are a source of error [79]. Thus basophilic stippling of itself should not be used to screen lead workers but only as confirmatory evidence when coupled with other tests. Another test which also proved to be equivocal in active plumbism was the determination of adrenal cortical function following the administration of ACTH [80]. In Europe, renal function tests including the determination of urea and creatinine clearance rates have been shown to be of value in detecting renal involvement in acute and chronic plumbism [81,82]. The limited number of cases of renal involvement in plumbism in the United States has not caused renal function tests to be used, but in the future, application of the radioisotopic renogram may prove of value.

The most valuable diagnostic aid in plumbism is the determination of blood and urine lead levels, but all samples must be collected under conditions that assure their freedom from outside contamination [83]. Analyses can employ spectrophotometric, spectrographic, polarographic, or atomic absorption procedures; however, only laboratories that make

frequent lead determinations can be considered trustworthy, and the data must be checked for mathematical accuracy [35,55]. Blood lead values above 80 μg/100 ml and urinary lead values above 200 μg/liter are indicative of active plumbism and require immediate action to prevent dangerous sequelae [6].

By its interference with heme synthesis at the Δ-aminolevulinic acid step, lead causes an increased excretion of ALA in the urine and this has been used as a method for detection of active plumbism [1]. It has been shown that urinary ALA increases in manifest as well as latent plumbism and can be considered the most sensitive test for lead exposure [84,85]. Furthermore, increased urinary ALA has been demonstrated for periods up to three years after the last known exposure to lead [86]. Moreover, the urinary ALA test is more sensitive than the urinary coproporphyria test for surveillance of lead-exposed individuals [87]; increased urinary ALA excretion only occurs in plumbism and acute intermittent porphyria, whereas coproporphyria excretion is increased in plumbism, liver disease, and acute alcoholism. In itself, the ALA test may not be conslusive but when combined with other diagnostic tests will give greater assurance of a valid diagnosis of plumbism [88,89]. The ALA test has been applied to traffic policemen in an attempt to ascertain whether air-borne lead from automobile exhaust was causing preclinical plumbism. It was concluded that such exposure had no effect [3].

The coproporphyrin test is one of the most widely used indications of lead overexposure. It has been employed as a screening test to determine the degree of exposure of maternity patients using lead-contaminated drinking water [90]. Even though increased urinary coproporphyrin is not specific for lead poisoning, any such increase should be investigated thoroughly before eliminating lead as the causative agent [91]. Russian workers are subjected to periodic coproporphyrin tests because experience has shown that this test indicated both early exposure and the stage of lead intoxication [6]. The value of this test for screening personnel for lead exposure in a steel plant has been amply demonstrated over a 15-year period when no overt exposures leading to active plumbism were allowed to occur [92]. Under any circumstances, a urinary coproporphyrin level above 800 μg/liter should be cause for immediate concern, and the entire battery of tests for plumbism used to determine the exact cause for such abnormal excretion [6].

Another test of diagnostic value is the edetate lead mobilization test coupled with the determination of both ALA and coproporphyrin. After the administration of 3 gm of edetate, urinary lead increased from 393 to

3299 μg/liter, ALA increased from 0.16 mg to 4.2 mg/100 ml, and coproporphyrin decreased [93]. The use of this test in France has materially reduced the need for job transfers and decreased the risk of lead storage in the body [7]. It also has been suggested that the edetate test may be useful in assessing lead absorption in industrially and nonindustrially exposed individuals [94]. There is some disagreement in how the lead excretion in this test should be expressed; some investigators use mg/liter and others use mg/24 hr. The latter feel that until the relationship between diuresis and lead excretion is clarified, only mg/24 hr gives valid results. Moreover, any lead excretion exceeding 0.35 mg/24 hr indicates close contact with lead and should be investigated [95]. Romanian experience with this test in 260 workers showed that it was helpful in cases where other diagnostic aid had failed [96]. It has also been suggested that the mobilization test is useful for diagnosis of lead deposits in the body [97]. Regardless of any disagreements concerning this test, it will continue to be used as a diagnostic aid.

The widespread use of edetate prophylactically in Europe indicates that a daily dose of 2 gm orally is effective in reducing the incidence of plumbism in industry [98-103]. However, it has been recommended that serum iron and copper levels be determined in any prophylactic program [103]. In the Philippines, edetate is administered prophylactically to only those workers showing a positive porphyrinuria [104]. On the other hand, prophylactic edetate should not be substituted for good industrial hygienic practices [105,106]; in fact, several workers have stated that such a procedure is senseless and unjustified [107,108]. American experience has also indicated that chemotherapy is no substitute for good industrial working conditions [35]. Prophylactic use of D-penicillamine is not as effective as edetate and in addition can cause severe allergic reactions [105].

Therapy

Although edetate is not recommended for prophylaxis, it has become the standard treatment for plumbism because it reduces body lead burdens in both soft and hard tissues [109]. This form of therapy varies in dosage from 0.6 to 2.4 gm daily for periods of 8 to 20 days; there is a marked reduction in both urinary lead and coproporphyrin at the termination of treatment. However, an increase in coproporphyrin usually occurs at this time due to mobilization of bone lead [110]. Spanish experience indicates that edetate therapy has a greater effect in counteracting peripheral nerve involvement than in correcting severe encephalopathic lesions [111].

In the absence of severe neurological involvement, edetate intravenously is very effective [112]. However, caution must be exercised, particularly in renal disease, because small amounts of edetate deposit temporarily in the renal basement membrane and can remove essential trace elements and cause renal damage [113]. In those instances where the patient requires quick deleading, or has renal impairment with azotemia, or where large doses of edetate may be hazardous, combination of peritoneal dialysis with edetate reduced the deleading time by one-quarter to one-third [114]. Comparison or oral versus intravenous administration of edetate indicated that the former was much less effective [115,116]. Moreover, only 5 to 10% of the oral dose is absorbed and doses above 3 gm cause nausea, abdominal distress, and diarrhea [117]. Comparison has also been made between the therapeutic effectiveness of edetate and desferrioxamine in deleading, and it was concluded that the latter was of doubtful value [118]. For the present and until better chelation agents are available, it appears that edetate will continue to be the most useful deleading agent in general use [119].

Another chelation agent which has been tried in plumbism is penicillamine. It appears to be as effective as oral edetate [120] and has the advantage of oral administration, but can cause sensitization [121]. The side effects include absence of reticular iron, exanthoma with itching, frequent eosinophilia, agranulocytosis, and granulocytopenia [122]. Some investigators have found penicillamine less effective in removing bone lead and have concluded that it offers no advantages over edetate [119,123,124]. Penicillamine also can produce a nephroxoticity not seen with edetate [125]. On the other hand, a direct comparison of edetate and penicillamine by both oral and intravenous routes of administration has led to the conclusion that intravenous edetate was more effective in increasing urinary lead than penicillamine; but the latter was more effective by the oral route and had the advantage that the lead complex was not resorbed in the gastrointestinal tract [126]. A comparison of the effectiveness of edetate, calcium trisodium pentetate (DTPA), and penicillamine showed that the last-named drug is 2 to 3 times less effective than either of the others [127]. This leaves the status of penicillamine as a deleading agent as still unproven.

Another deleading agent, in use in Europe but not in the United States, is calcium trisodium pentetate (DTPA). The drug has the advantage that it can be given intramuscularly, and it is as effective as intravenous edetate [128]. Pentetate has the ability to chelate both iron and copper and in deleading, it has been shown to increase both urinary iron and copper [129,130]. Another feature of pentetate is its lipotropic nature

which allows greater cell penetration. This may be a good property in the therapy of lead encephalopathy, but, on the other hand, might damage the islet cells of the pancreas causing diabetes [109]. More work must be done to establish the exact place of pentetate in the treatment of lead intoxication.

SUMMARY

There are numerous differences between the two forms of active plumbism. Pica and lead fumes are the usual cause of pediatric variety, whereas poor industrial hygienic practices produce adult plumbism. The pediatric disease involves hematological, gastrointestinal, and neurological symptoms, and the latter include drowsiness, coma, and grand mal seizures. Brain damage has implicated impedence of brain hemodynamics with changes in both white and gray matter. Adult plumbism requires more time for its development and presents a nonspecific symptom complex with low hemoglobin, gastrointestinal upsets and increased delta aminolevulinic acid and coproporphyrin in the urine. There may be basophilic stippling and lead line on the gums, but these indications should not be used as sole diagnostic aids. With further development of the disease, the neuromuscular involvement becomes pronounced and in those instances where females are involved, sterility and abortion have been observed. The most valuable diagnostic aid is the determination of blood and urine lead levels, but only by a competent laboratory. The edetate lead mobilization test is also valuable. Therapy for pediatric plumbism includes intravenous edetate to remove blood lead and either urea or mannitol to reduce cerebral edema. Corticosteroids have also been used. In the adult form of the disease, edetate is also effected and in its absence, penicillamine can be used. Pentetate has been used in Europe, but its damaging effects on the islet cells of the pancreas may make its use questionable.

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