Neurologic Sequelae of Plumbism in Children

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Prevention is still the best treatment for lead poisoning and its sequelae.

Here is a survey on the nature and incidence of the neurologic sequelae of lead intoxication among 425 patients over a ten-year period.

LEAD has become a prime cause of poisoning among children in the United States, particularly in the lower socio-economic group. In and around Chicago, lead accounts for nearly 80 per cent of all deaths due to accidental poisoning.[4] Intoxication from arsenic, thallium, salicylates, and other chemicals usually result in either death or complete recovery, but plumbism is far more likely to leave permanent sequelae.

Mortality rates and the incidence of sequelae from lead poisoning vary both with the age of the patient and the clinical picture at the onset of the disease. They are greater among infants and among those presenting with severe encephalitic symptoms, than among older children and those who present with only gastro-intestinal symptoms or general malaise without neurologic symptoms. Morbidity and the incidence of sequelae are not necessarily related to blood lead or urinary coproporphyrin levels, either, since some of the most severely involved patients may have only modest increases in blood lead and urinary coproporphyrins at the time of admission.

The clinical aspects and mortality in lead poisoning in the Chicago area have been reviewed elsewhere.[4-7] The purpose of this paper is to delineate and to describe the nature and incidence of the neurologic sequelae in those patients who survive. The sequelae of lead poisoning have been documented by Byers and Lord[1, 2] in 20 children, by Thurston *et al*.[11,12] in 11, by Smith[10] in 30, and by Chisolm and Harrison[8] in 36 children. The most common sequelae reported include mental retardation, recurrent seizures, cerebral palsy, and optic atrophy. Although others have reported sequelae involving kidney, liver, heart, and muscle, these are beyond the scope of this present review.

As in all retrospective studies, the condition of the children in this study before the onset of plumbism has to be surmised from the history. Thus, one cannot always be sure that some of the observed sequelae do not antedate the plumbism. Children who are mentally retarded are more likely to indulge in pica, and children who develop plumbism from pica are more likely to become mentally retarded.

Plan of Study and Materials

Of 425 patients in this study, 389 were from the Children's Neurology Clinic of the Cook County Hospital, and 36 from the private practice of the senior author. Most of the Cook County Hospital patients had been initially hospitalized, and on discharge had been followed through the Out-Patient Clinic for periods ranging from six months to ten years. Eighty-four per cent were Negro children from the slum areas in Chicago. There were 214 male and 211 female patients and their ages ranged from nine months to eight years at the time of the first admission, with the median age around two years—the age at which most accidental poisonings occur.

The source of lead in 408 patients was from chewing or eating loose plaster with old lead-containing paint on it in slum clearance projects; the burning of battery casings was responsible for intoxication by the inhalation of lead in 17 instances. In a pair of twins, the lead source was from sucking on lead-containing toys, and in one case, from water in a tank contaminated by red

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lead paint. Case distribution and sources of lead are summarized in Table 1.

The histories were verified, when feasible, by social service interviews, and the pre-plumbism status of the patients was judged by detailed developmental histories or by school records when available. The incidence of each type of sequela was correlated with the mode of clinical onset and the source of lead exposure.

Results of Study

In 257 or 61 per cent of the 425 patients studied, there was complete recovery from lead intoxication; in 168 or 39 per cent, neurologic sequelae remained, and these are summarized in Table 2.

Sequelae of Lead Poisoning

The sequelae were grouped into four main subdivisions:

(1) *Mental retardation* was the most common sequela, occurring in 22 per cent of the total series. Included in this group were some children who had had pre-existing mental retardation as judged from the developmental history or I.Q. records when available, but in whom the intellectual deficit was aggravated as a result of the plumbism.

The degree of retardation was usually profound. In a few patients, the intellectual involvement was less marked, and I.Q. scores were in the low normal category. These were included only if their I.Q. had been average or above before the plumbism. In some, the brain damage was minimal with learning blocks, usually of a visual-perceptual type, being the only sign of impaired learning.

TABLE 1. *Distribution of 425 Cases of Plumbism*

Area		No. of Patients
Source	Cook County Hospital	389
	Private Practice	36
Race	Negro	357
	White	68
Sex	Male	214
	Female	211
Age	9 mos. to 8 yrs. (Median age 2 yrs.)	
Source of Lead		
Enteric		
	Pica for paint or plaster	405
	Lead toys	2
	Contaminated water supply	1
Respiratory		
	Burning of battery casings	17

(2) *Recurrent seizures* were almost as common, occurring in 20 per cent of the patients. Only those who had previously been seizure-free, or whose pre-existing seizures had been perceptibly aggravated by plumbism were included in this group. The most common type of seizure was grand mal, present in 85 per cent, although focal, Jacksonian, myoclinic, akinetic and vegetative spells often co-existed with the predominant grand mal seizures. Only 10 per cent of the patients with seizures had focal or Jacksonian convulsions without accompanying grand mal. In only three patients did myoclonic, akinetic, and

TABLE 2. *Sequelae of Plumbism by Mode of Onset in 425 Patients*

	Mode of Onset															
	Total (N = 425)		Enceph. (N = 59)		Seizures (N = 43)		Ataxia (N = 17)		G.I. (N = 232)		Febrile (N = 16)		Asymptomatic (N = 58)	
Sequelae	*N*	%	*N*	%	*N*	%	*N*	%	*N*	%	*N*	%	*N*	%
None	257	61	11	18	14	33	7	41	159	69	13	81	53	91
Mental retardation	93	22	23	38	14	33	5	29	43	19	3	19	5	9
Seizures	85	20	32	54	17	39	6	35	30	13	0	0	0	0
Cerebral palsy	9	2	8	13	0	0	1	6	0	0	0	0	0	0
Optic atrophy	5	1	4	6	0	0	1	6	0	0	0	0	0	0
Total		106%*		123%*		105%*		117%*		101%*		100%		100%

* Percentages total more than 100% because of multiple sequelae in same patient.

vegetative seizures, respectively, each occur as the only type of residual seizure.

(3) *Cerebral palsy* was third in frequency as a sequela, occurring in only nine patients or 2 per cent. Spasticity was the most common type, present in six children, five hemiplegics and one triplegic. Extrapyramidal forms of cerebral palsy occurred in three patients—one ataxic, one with combined athetosis and hemirigidity, and one with an unusual complication of progressive dystonia musculorum deformans. Case reports of three of these patients are appended.

(4) *Optic atrophy* was the most common ocular sequela, occurring in five patients or 1 per cent. One of these also had retrobulbar neuritis, one had corneal opacities, and one had bilateral ptosis. Strabismus occurred in three other patients.

Mode of Onset of Symptoms

From a prognostic viewpoint, it is interesting to correlate the incidence and types of sequelae with the mode of onset of the disease. For purposes of this study, the mode of onset was divided into six clinical types, three with neurologic symptoms, and three without.

1. *Encephalopathic.* These patients had the most severe neurologic involvement. There was usually an acute or subacute onset with signs of increased intracranial pressure—clouded sensorium, slowing of the pulse or respiration, choked discs, and increased cerebrospinal fluid pressure. Coma, fever, ataxia, convulsions, and cerebrospinal fluid pleocytosis and increased protein were also usually, but not always present on admission or a few days later.

2. *Seizure.* Here, the presenting symptom was a seizure in a child previously seizure-free. The seizure was generally a grand mal, focal, or vegetative spell, with or without the presence of fever. In some of these patients, ataxia of variable degree did co-exist, but the signs of increased intracranial pressure, characteristic of the encephalopathic group, were absent.

3. *Ataxia.* In these patients, ataxia was the only or primary presenting neurologic sign. The onset was generally less acute or less dramatic than in the two preceding groups. There was usually a gradually progressive ataxic gait plus incoordination in hand function, with or without nystagmus. Of the three neurologic types of onset, the ataxic group was the most benign.

4. *Gastro-intestinal.* These patients presented not with neurologic symptoms, but with subacute or chronic complaints of nausea, vomiting, colic, constipation, or diarrhea. There might also have been some evidence of anemia, malnutrition or general malaise, but gastro-intestinal complaints predominated. Children entering the Cook County Hospital with such complaints are viewed with suspicion of lead poisoning unless proven otherwise.

5. *Febrile.* The onset here was not suggestive of plumbism. The presenting symptom was fever, with or without signs of infection. In many of these patients, the acidosis associated with the febrile episode with its mobilization of stored lead might have been responsible for the later superimposition of secondary gastro-intestinal or neurologic symptoms.

6. *Asymptomatic or subclinical.* In these children, the diagnosis of plumbism was made either by our routine workup of asymptomatic siblings of children with known lead poisoning, or was an inadvertent or accidental finding.

Incidence of Sequelae Classified by Mode of Onset

As expected, mortality, morbidity, and all types of sequelae were most frequent in patients with an encephalopathic onset of symptoms. In this group, the mortality varied from 28 per cent to 45 per cent [5] depending upon the treatment employed and being greatest among those in whom extensive craniotomies were done. The incidence and nature of the sequelae, however, was not essentially different with both types of treatment.

In 59 patients surviving encephalopathy and followed for six months to ten years, sequelae, summarized in Table 2, persisted in over four out of five or 82 per cent. The most common sequela was recurrent seizures. These occurred in over one-half or 54 per cent of the patients, were generally resistant to drug control, and were of a mixed type, such as a combination of grand mal, focal and myoclonic.

Mental retardation was the next most common sequela, being present in 38 per cent. It was generally of a profound type and many of these patients remained vegetative. Cerebral palsy occurred in only 13 per cent of these patients, but it is interesting that of the nine in the total series who were left with cerebral palsy, eight presented with encephalopathy. Mental retardation and seizures co-existed in many of these cases.

Optic atrophy, the least common sequela, occurred in only four or 6 per cent of the patients. However, of the five patients with this complication, four were encephalopathic on first presentation. This relatively low incidence of optic atrophy does not necessarily reflect a decreased sensitivity of the optic nerves to lead, but, rather, that this sequela occurs only in severe cases, most of whom die. In the patients with optic atrophy, one had

a concomitant retrobulbar neuritis, another had corneal opacities, and a third had ptosis.

In some patients there were multiple sequelae, so that a total of 67 sequelae were recorded in 59 patients.

Of 43 patients presenting first with seizures, but with no other evidence of encephalopathy, 14 or 33 per cent had no sequelae. Among the 29 with sequelae, recurrent seizures were most common and persisted in 17 patients or 39 per cent. These seizures, primarily of the grand mal type, were generally more amenable to drug therapy than seizures following the encephalopathic onset of lead intoxication. Mental retardation occurred in 14 or 33 per cent of patients, two of them with recurrent seizures. The mental retardation was generally less severe than in the encephalitic group. There was no cerebral palsy or optic atrophy in this category.

Among 17 patients, the onset of symptoms was with ataxia alone. There were no seizures or increased intracranial pressure. In seven or 41 per cent, there were no sequelae. Six or 35 per cent developed recurrent seizures, five or 29 per cent were left with mental retardation, and one with optic atrophy and ptosis. One other patient who developed progressive dystonia musculorum deformans, and described in Case Report No. 3, had repeated exposures to lead and may have had an unrecognized encephalopathy.

In three patients who presented with ataxia, combined sequelae existed; the patient with optic atrophy also had seizures, and two of those with seizures also became mentally retarded. It is interesting that in no patient presenting with ataxia alone did this symptom persist. The only patient in the total series in whom ataxia remained as a sequela had an encephalopathy associated with seizures and ataxia.

The largest group of patients, 232, presented with gastro-intestinal symptoms, such as nausea, vomiting, constipation, diarrhea, and colic. In this group, sequelae were relatively infrequent, occurring in only 31 per cent. In 30 or 13 per cent of these patients, seizures persisted. These were mainly of the grand mal type, and most were amenable to drug therapy. Mental retardation remained in 43 or 19 per cent. It was less profound than that in the group with encephalopathic or seizure onset. Four patients with a severe degree of mental retardation actually had aggravation of a pre-existing intellectual defect, one of them due to meningitis at one year of age. There was no cerebral palsy or optic atrophy as sequelae in this group. Of four patients with sickle cell anemia, none developed sequelae.

The group which presented with fever was the smallest. It consisted of 16 patients who presented with fever only, with or without an associated infection. As expected with this relatively benign type of onset, sequelae were less frequent, even though transitory neurologic symptoms may have appeared in the course of the febrile episode. The only sequela was mental retardation, and this was seen in only three or 19 of these patients. Among these three, intelligence was definitely less than before the febrile episode, but whether the mental retardation was attributable to plumbism or to some other infective factor is not certain.

Among the asymptomatic patients, numbering 58, no symptoms referable to plumbism were present. The diagnosis was made accidentally, or because a sibling was known to have plumbism. In one family where ten patients were examined because a sibling had lead encephalopathy from the burning of battery casings, no sequelae were found. In another where two children had died from lead encephalopathy, five siblings were followed for two years. Of these, three were perfectly normal, one had an I.Q. of 85 not attributable to the lead exposure, and one had seizures before the lead exposure which were not aggravated after it. In another family where five siblings were followed for 18 months, two children were perfectly normal and three showed slightly retarded to low normal I.Q.'s, but were considered to have familial or socio-cultural type of retardation, rather than that due to plumbism.

In only five patients or 9 per cent of those who were asymptomatic on first presentation was mental retardation considered to be a possible sequela. Even in these, however, the reliability of the maternal histories was questionable. In only one of these had the onset of mental retardation been suddenly noted when a sibling developed lead encephalopathy.

Sequelae Classified by Origin of Plumbism

No significant differences in the incidence or nature of sequelae were found among children in whom the disease followed pica and among those from lead vapor inhalation. Re-exposure or multiple exposures to lead, however, did tend to result in greater mortality, morbidity and sequelae.

Significant Case Reports

In an attempt to better describe the treatment and outcome of our cases of lead intoxication, we have included here three of our most interesting cases in detail.

1. *Lead encephalopathy with left spastic hemiplegia, focal seizures, mental retardation, and optic atrophy as sequelae.*

S. D. was a two-year-old Negro girl who was admitted on July 28, 1962 with history of anorexia for six months, vomiting for two weeks, and sore throat, and listlessness for two days. The patient had been picking paint off the window sills. She lived with two older siblings and a 24-year-old unmarried mother on relief in a two-room dilapidated apartment. Except for a birthweight of 3 lb., the pregnancy, delivery, neonatal period and development were normal. The patient had walked at 11 months and spoken sentences at two years.

On admission, she was lethargic, undernourished, pale, and dehydrated. Except for slight nuchal rigidity and mild papilledema, the physical examination was normal. Cerebrospinal fluid was clear, but under increased pressure—600 mm. of water opening and 300 mm. closing pressure; there were 6 lymphocytes, 90 mg. glucose, 78 mg. protein, and 109 mg. chloride per cc. Urine coproporphyrin was 4+. X-rays showed dense epiphyseal lines in the long bones and radio opaque material in the bowel. There was a mild anemia with hypochromia and basophilic stippling of the red blood cells. The blood lead was 140 micrograms per 100 ml. A diagnosis of early lead encephalopathy was made.

After cleansing enemas to remove rectal and colonic lead, intravenous urea, 8 Gm., and methylprednisolone succinate, 6 mg. q 8 hr., were started. On the third day, the patient became irritable, and began to have generalized seizures controlled by intravenous pentobarbital sodium, ether inhalation, and rectal paraldehyde. Aspiration of vomitus on one occasion was followed by respiratory arrest; this was relieved by tracheostomy and positive pressure respirator. The patient remained comatose and pupils reacted sluggishly to light. On the fourth day, Calcium Versenate, 300 mg. b.i.d., was started intravenously and given for five days. On the fifth hospital day, because of rising temperature and poor condition, hypothermia was induced to 94° F. rectally.

From the fifth to eighth days, twitching of the left side, generalized seizures, and multiple premature heart beats with sinus arrhythmia were observed, but the blood pressure, urinary output, and serum electrolytes remained normal. On the ninth day, the patient was more responsive and opened her eyes; by the 11th day, the tracheostomy was removed. It was now noted that the left arm was flaccid and the left knee jerk exaggerated. Urine coproporphyrin was still 3+ on the 20th day. On the 30th day, after four more five-day courses of Versenate and eight courses of urea, she was walking with help, but with an unsteady gait. She was discharged and referred to the clinic where she was followed for the next 18 months.

The ataxia disappeared within two months, but focal and generalized seizures persisted; there was a mild residual left spastic hemiplegia. When last seen bilateral optic atrophy and profound mental retardation to the idiot level were present. Speech had not been regained, and patient still needed minimal support for walking.

Comment: The encephalopathy in this child was not aborted by urea, steroids, hypothermia, or Versenate. The brain damage may have been compounded in some measure by the respiratory arrest. The long exposure to lead and the relative youth of the patient were probably the most important factors.

2. *Lead encephalopathy and duodenal ulcer from pica with residual ataxia, seizures, mental retardation, and optic atrophy.*

W. G., a 29-month-old Negro boy, was admitted on August 5, 1962 with a history of plaster pica for several weeks, vomiting, constipation and lethargy for nine days, and fever for five days. He had previously been normal; he walked at 11 months, spoke words at nine months, and used sentences at 14 months.

On admission, except for lethargy and hyperirritability, the physical and neurologic findings, including fundoscopic, were normal. Spinal tap showed 600 mm. of water opening, and 560 mm. closing pressure. A Pandy test for spinal fluid globulin was positive, and there were 60 lymphocytes, 98 mg.% glucose, and 204 mg.% protein per cc. Urine coproporphyrin was 4+. X-rays showed typical heavy epiphyseal lines in the long bones, radio-opaque material in the colon and rectum, and some separation of the skull sutures. There was a secondary anemia, but no basophilic stippling. The blood lead was later reported as 110 micrograms per 100 ml. A diagnosis of early lead encephalopathy was made.

Cleansing enemas were given, followed by intravenous urea, 14.2 Gm. q 8 hr., and methylprednisolone succinate, 8 mg. q 6 hr. There was slight improvement after the second course of urea, but on the second day, the patient became more irritable and apprehensive, his neck became rigid, and by the third day, he started to convulse. Convulsions became progressively worse and were only partially controlled with sodium amytal. Papilledema and deepening coma were now noted. Vital signs remained stable. Urea was continued and temperature was reduced to 94° F. rectally by ice-water mattress. On the sixth day, while still convulsing, he had a tarry stool and stopped breathing. Tracheostomy was performed and a positive pressure respirator attached after resuscitation.

Calcium Versenate was started intramuscularly on the seventh day, 540 mg. b.i.d., and given for five days. This was repeated until three courses were given. Bleeding from the bowel continued, and the hematocrit dropped from 32 to 16. A whole blood transfusion was given and repeated on the eighth day. Urinary coproporphyrin was still 3+ after the Versenate. On the 16th day, tarry stools still continued and the hematocrit again dropped to 15. Another transfusion was given and steroids were decreased.

Emesis and melena failed to respond to medical treatment, including gastric balloon insertion, and on the 25th day, a laparotomy was performed. A 1 cm. indurated chronic posterior duodenal ulcer was found, plus a small intestinal tear 4 inches distal to the ligament of Trietz, probably due to the balloon. A pyloroplasty, small bowel resection, and bilateral vagotomy were done. The procedure was well tolerated, bleeding stopped, and the patient began to improve. By October 4, 1962, two months after admission, the patient had developed corneal opacities, poor vision, loss of speech, mental retardation, and an inability to walk. He was discharged and referred to the clinic.

First clinic visit was three months later. At this time, he had mild recurrent right focal seizures lasting two to three minutes, becoming generalized. These were eventually controlled with Dilantin and Mysoline. Ability to walk alone returned six months after discharge, but still with ataxia. There was a bilateral severe optic atrophy, bilateral corneal opacities, and almost no reaction to visual stimuli. In April 1965, two and one-half years later, the patient still did not talk, was incontinent, could not feed himself, and was profoundly retarded. He was still walking with an ataxic gait. No involvement of the eighth nerve could be demonstrated.

Comment: The severe neurologic sequelae may have been due in part to the anemia, respiratory arrest, and surgery. Ataxia of central (presumably cerebellar) origin is one of the less common permanent sequelae of plumbism. The complication of a bleeding duodenal ulcer is of interest, and not too uncommon.

3. *Onset of plumbism with ataxia, tremors, and later extrapyramidal rigidity, but without signs of increased intracranial pressure. The course continued as a progressive dystonia musculorum deformans, a rare complication.*

A three-year-old Negro boy, previously well, was admitted on June 5, 1958 with a history of vomiting and weight loss for five weeks, unsteady gait for three weeks, and fever for one day. A two-year history of pica was elicited. The patient was a full-term normal infant of birthweight 8 lb. 5 oz. He had walked at 11 months and spoken sentences at 18 months.

On admission, there was exudative tonsilitis for which penicillin was given. Pulse, respiration, temperature, and blood pressure were normal. The only abnormal neurologic finding was an ataxic gait plus coarse tremors of the hands, especially on intention. There was no increased intracranial pressure; spinal tap and fundoscopy were normal. X-rays of the bones showed typical lead lines, and urine coproporphyrin was 4+. The blood count showed a moderate anemia. Blood lead level was 85 micrograms per 100 ml.

The patient was given two five-day courses of intramuscular Calcium Versenate, 400 mg. b.i.d. He also received oral Calcium Versenate, 500 mg. q.i.d. for ten days. By the 12th day, the urine coproporphyrin had disappeared, and the gait had improved. He still had some ataxia and tremors, however, when discharged three weeks after admission. Four weeks later, August 2, 1958, he was readmitted because of vomiting for four days and a gait which was becoming more unsteady since discharge from the hospital. Pica for plaster had persisted since discharge.

On this second admission, the patient was still ambulatory. General examination was normal except for slight pallor. Neurologic examination revealed a propulsion gait, with tendency to fall to the left. Tremors and cogwheel rigidity were present bilaterally, more on the left. Urinalysis showed 4+ coproporphyrin. The hemoglobin was 6.9 g. per 100 ml. (45%); RBC's numbered 2.38 millions; Turk irritation cells were seen in the differential, but there was no basophilic stippling of the red blood cells. There was no evidence of increased intracranial pressure or cerebrospinal fluid change noted on lumbar puncture.

The patient was treated again with Calcium Versenate for five days, 250 mg. b.i.d., and was placed on iron supplements. A therapeutic trial with Artane for two weeks to control tremors and rigidity was without benefit. The patient was discharged after eight days still walking with an unsteady gait to be followed in the Pediatric Neurology Clinic.

His urine coproporphyrin and blood lead normalized, and the lead lines decreased over the course of six months. On the Stanford Binet, his I.Q. in 1959, at a chronologic age of four years two months was 74, and in 1961, at a chronologic age of six years four months, it was 70. This was considered to be an intelligence loss from his presumably average pre-plumbism intelligence, as judged from his developmental history. Speech was distinct and hearing adequate for ordinary conversation.

In spite of no further lead ingestion, however, the boy's motor condition became gradually worse. He developed extraneous involuntary movements which began in the left leg and then spread after a few months to all four extremities. These movements were dystonic and more marked in proximal and trunk muscles. While walking, his back would suddenly arch and his leg become forcibly flexed at the hip and knee, and he would frequently fall. He gradually began to assume the bizarre dystonic postures seen in typical dystonia musculorum deformans. The child finally became wheelchair and bedridden.

During all this period, his speech and mentality, although reduced from his presumably pre-lead average levels, seemed to be adequate for normal use. When last seen, in April, 1966, nearly eight years after his first admission, a trial on Valium was given with moderate improvement in his muscle tension. A chemopallidectomy was being considered as the next possible procedure to relieve his deforming postural movements.

Comments: A double exposure to lead is seen here, the first being followed by cerebellar ataxia, and the second by extrapyramidal rigidity becoming progressively worse and developing dystonic athetosis. To our knowledge, the clinical syndrome of a progressive dystonia musculorum deformans as a sequela of plumbism is unique.

The absence of signs of increased intracranial pressure is of interest. It may be speculated that the first episode of plumbism caused a "locus resistentia minoris" in the central nervous system, so that the brain was more vulnerable to the continued exposure to lead.

Comments and Discussion

Mortality and sequelae of lead poisoning in children are still very high. The more fulminant the onset of plumbism and the younger the child at onset, the greater are the mortality and the incidence of sequelae. In adults, the neurologic sequelae are primarily of a peripheral neuropathic nature, characteristically with wrist drop due to radial palsy. In children, the central nervous system is more likely to be involved, since the younger the child, the more vulnerable the brain to lead intoxication.

The greater central nervous system involvement may also be due in part, as sugested by Chisolm and Harrison,[8] to the more massive and acute exposure that occurs in childhood. Since lead encephalopathy is more common in children than in adults and since more vital centers are involved in youngsters, it is understandable that mortality and morbidity from plumbism is higher in children.

In childhood lead encephalopathy, the mortality is between 28 per cent and 45 per cent, and four out of five who survive have sequelae. The mortality and sequelae are decreased when the onset is less acute, even though the presenting symptom may be a neurologic one, such as seizures or ataxia. In the most commonly seen form of plumbism, that which presents with gastro-intestinal symptoms, the mortality is negligible, and only three out of ten patients have sequelae.

Mental retardation and recurring seizures, the most common sequelae seen in lead poisoning, reflect the diffuse severe cortical involvement.[8, 11] The degree of mental retardation is variable and dependent upon the severity of the disease. In the mildly involved, there are often only perceptual problems, primarily of a visual type. Seizures caused by plumbism, as is true of most seizures due to organic brain involvement, are more refactory to anticonvulsant therapy than idiopathic epilepsy.

The cerebral palsies were most commonly seen in children presenting with encephalopathies. As in most acquired cerebral palsies,[9] these tend to be primarily of the spastic variety. The greater susceptibility of the "pyramidal tract system" in infancy may be due to its relatively incomplete myelinization and development, as compared to the basal nuclear or extrapyramidal areas, whose injury might result in dyskinetic syndromes.

In only one instance was the ataxic form of cerebral palsy a sequel, in spite of the fact that ataxia was not uncommon as a presenting symptom. The occurrence in one patient of dystonia musculorum deformans progressiva, described in Case Report 3, after repeated exposures is unique in our experience. Optic atrophy with or without neuritis was the most common cranial nerve symptom. Ptosis, corneal opacities, and strabismus occasionally occurred. In our experience, deafness due to involvement of the eighth nerve did not occur.

Although the fatality rate was greatest in those treated by cranial decompressions, the incidence and nature of sequelae was not related to the type of treatment of the encephalopathy, or to the portal of lead entry, whether by ingestion or by inhalation. Re-exposure and continuing exposure to lead, however, increases mortality and morbidity. As a result of re-exposure, the subclinical case may begin to develop gastro-intestinal and then neurological symptoms. The mild case may develop ataxia, then seizures, and finally, a fulminant encephalopathy.

Other factors which may trigger a latent plumbism into an active form may be of a metabolic or seasonal nature. Acidosis, incident to infections, malnutrition or to increased ultraviolet radiation as occurs in summer, may precipitate an acute episode of plumbism. Thus, it becomes important to detect plumbism in the early stages, before irreversible neurologic damage can occur. Once neurologic systems have become involved, they may become sensitized target organs vulnerable to repeated or continuing lead ingestion. At the present time, the best treatment for lead poisoning and its sequelae is its prevention.

Summary of Findings

Among 425 children with plumbism, 39 per cent had neurologic sequelae. Mental retardation and recurrent seizures are most common and persist in approximately one out of five patients. Cerebral palsy and optic atrophy occur less frequently and are limited to those who present with either encephalopathic or ataxic syndromes.

Cerebral palsy is usually of the spastic hemiplegic type. A patient who developed dystonia musculorum deformans is unique in our experience. The younger the child and the more fulminating the onset, the greater the incidence of sequelae.

In children presenting with severe encephalopathy, sequelae persist in over four out of five. In those who present with seizures without increased intracranial pressure, sequelae occur in two out of three. When ataxia is the only neurologic presenting symptom, sequelae occur in three out of five. In those in whom the presenting symptoms are gastro-intestinal, sequelae occur in less than one out of three. When the onset is with a fever only, or asymptomatic, the incidence of sequelae is one out of five to ten.

The nature and incidence of sequelae seems to be unrelated to the type of treatment employed.

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