American Journal of Diseases of Children

VOLUME 66 NOVEMBER 1943 NUMBER 5

LATE EFFECTS OF LEAD POISONING ON MENTAL DEVELOPMENT

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FOREWORD

The sudden tragic death of Dr. Lord on Jan. 10, 1943 occurred before the completion of the final draft of this paper. It was she who first felt that the minor deviations found on psychologic examination of these children with lead poisoning might be of important significance for the future. It was she who maintained the file of cases, studying them from the psychologic point of view, without encouragement from any one for the first several years.

That lead poisoning occurring in early life usually has a disastrous effect on mental development has not been generally recognized, though the subject of lead poisoning in children has been discussed by many observers. The manifestations of acute involvement of the nervous system have been adequately described, and the gross destructive lesions in the brain consequent to acute lead encephalopathy accompanied by cerebral edema and high intracranial pressure have been recognized. On the other hand, McKhann,[1] for instance, stated: "The neurologic manifestations of lead poisoning usually subside without serious consequences if the ingestion of lead is stopped and the removal of lead from the circulation and its deposition in inert form in the bones can be hastened, as described, by the use of a diet high in calcium together with the administration of cod liver oil or viosterol to accelerate the laying down of new bone."

This point of view, which is pretty generally accepted, fails to take into account the effect of lead poisoning on the growth and development of the infantile nervous system. The present observations, in our opinion, demonstrate that this process may be seriously impaired, showing as they do that of 20 children with mild lead poisoning in infancy, only 1 has progressed satisfactorily in school.

The primary requisite for supporting such a contention is the proof of early lead poisoning. Aub, Minot, Fairhall and Reznikoff [2] in 1925 published a monograph on lead poisoning and in it outlined standards for establishing the diagnosis based on earlier suggestions of Newman, McConnell, Spencer and Phillips. While criticisms of such an outline were undoubtedly valid, it may serve as a concrete basis for discussion. Of first importance was the recognition of a source of ingestion or inhalation of lead. In addition, at least two symptoms from the following list were required: marked pallor or anemia, colic or obstinate constipation, muscular incoordination, peripheral motor paralysis of the most used muscles

Mrs. Penfield Roberts gave valuable assistance in summarizing Dr. Lord's notes on some of the psychologic examinations.

Dr. Lord's work on this paper was done under a grant by the Earhart Foundation, and Dr. Byers' work, under a grant by the Commonwealth Fund.

1. McKhann, C. F.: Lead Poisoning in Children: Cerebral Manifestations, Arch. Neurol. & Psychiat. **27**:294 (Feb.) 1932.

2. Aub, J. C.; Fairhall, L. T.; Minot, A. S., and Reznikoff, P.: Medicine **4**:1, 1925.

(usually the dorsiflexors of the feet in children), basophilic stippling of the red cells, a lead line on the gums and lead in abnormal quantities in the stools and urine. If two of the foregoing symptoms were lacking, a presumption of lead poisoning could be established by the presence of three of a longer list of less specific symptoms, among which were emaciation, loss of appetite, especially for breakfast, vomiting on eating solid food, abdominal pain, loss of strength, headache, insomnia, mental lethargy, tremor, dizziness, encephalopathy, hypertension and articular pains.

Since these suggestions were made, some new criteria for diagnosis have been developed. Vogt,[3] Park, Jackson and Kajdi,[4] Caffey [5] and Kraft and Kato,[6] independently and more or less simultaneously, published papers showing that in

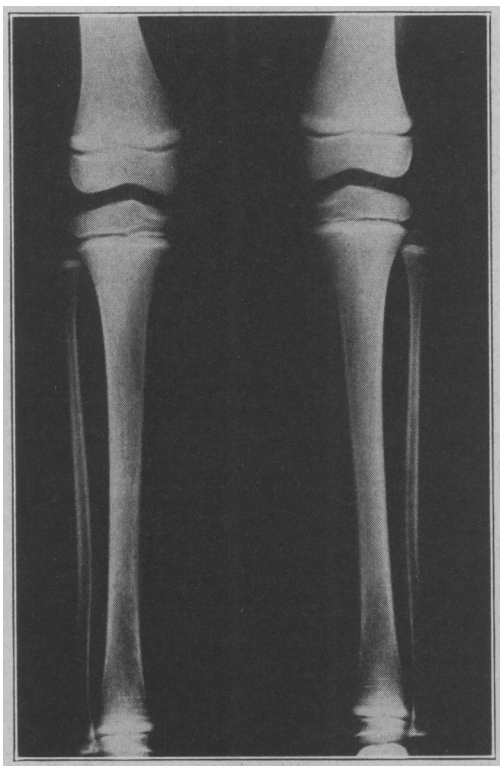

Fig. 1 (case 6).—Well marked deposits of lead at the diaphysial margins of a long bone.

children during the period of rapid growth while the process of lead ingestion was occurring lead was deposited in special concentration in abnormally dense bone at the growing ends of the shafts of the long bones and along the growing margins of the flat bones. These deposits formed a radio-opaque band, comparable in density to that of the lead numerals used to mark roentgenograms, and when well developed were very characteristic. When less well developed they could be simulated by the changes of rapidly healing rickets, by deposits of bismuth salts or possibly of other metals and, finally, by the dense bands resulting from the admin-

3. Vogt, C.: Radiology **22**:87, 1934.

4. Park, E. A.; Jackson, D., and Kajdi, L.: Shadows Produced by Lead in X-Ray Pictures of Growing Skeleton, Am. J. Dis. Child. **41**:485 (March) 1931.

5. Caffey, J.: Radiology **17**:957, 1931.

6. Kraft, E., and Kato, K.: Fortschr. a. d. Geb. d. Röntgenstrahlen **46**:249, 1932.

istration of elementary phosphorus in cod liver oil. Typical dense bands at the growing margins of the shafts of the long bones, in conjunction with a history of ingestion of lead, were considered by the various authors as reliably diagnostic of lead. Much of the roentgen evidence in the present series was seen and interpreted by Dr. Vogt.

Another roentgen sign rather nonspecific was the finding of flocculi of radio-opaque material in the intestinal tracts of children who had recently been chewing paint. It had been of clinical value mainly in calling to the attention of parents previously unobserved pica. It was, of course, much more useful in outpatient

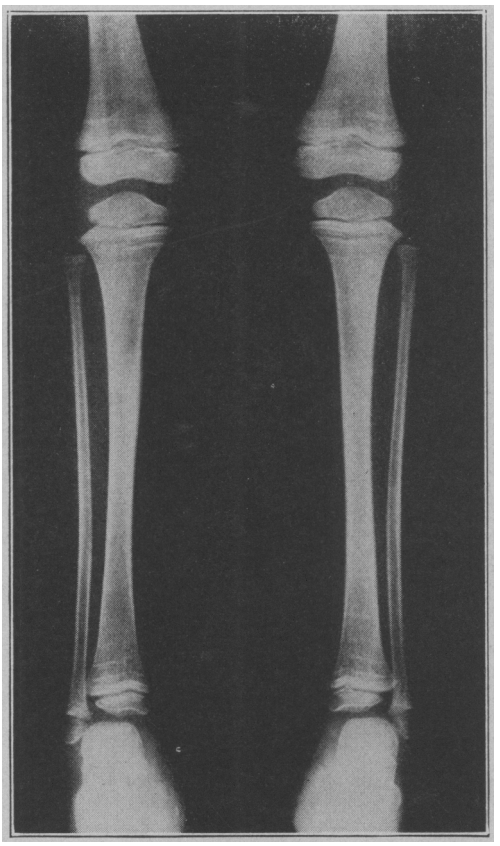

Fig. 2 (case 12).—Recurrent deposits of lead at the margins of the shafts of the long bones.

or office practice than in the hospital ward, where such evidence disappeared in a few days.

In relation to diagnosis, it was also clear from the review of Aub and his co-workers [2] that lead in large amount may be retained in the body, especially in the skeleton, in the absence of any of the clinical evidence of lead poisoning. Although this lead had been regarded as harmlessly stored in the skeleton, they showed that in animals free of symptoms of lead poisoning and protected for months from ingestion of lead small amounts of lead were excreted both by bowel and by kidney for a long time. Since practically all the lead in the bodies of human beings and of animals that had been exposed to lead and then protected from it for a considerable time was in the skeleton, such excretion presupposed

continual solution and transportation by the blood stream of small amounts of lead from the stores in the bones. The same authors also measured the urinary and fecal excretion of lead by patients recovered from lead poisoning and found it virtually uninterrupted for long periods. The skeletons of persons exposed to lead were found to contain from approximately 200 to approximately 900 mg. of lead, and the rate of combined excretion through bowel and kidney of the recovered patients varied between 0.07 and 0.89 mg. daily. Under these circumstances, it would take the person with the least lead about seven months to excrete at the most rapid rate all the lead stored in the skeleton. Since the person with the least lead was an infant who had chewed the paint from her crib "for only a few weeks," it seems likely that the amounts of lead involved in our cases were of the same order of magnitude. It must also be remembered that in a person with such recent exposure to lead, considerable amounts of lead were contained in organs

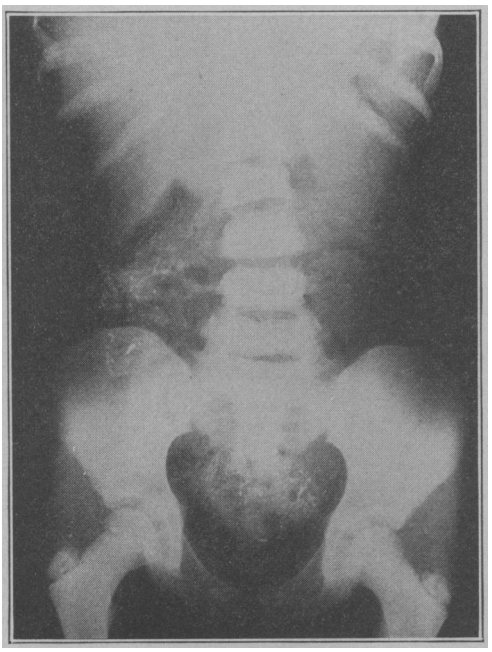

Fig. 3 (case 6).—Radio-opaque material in the intestinal tract, consistent in its appearance with that of flecks of paint.

other than the bones. In addition, the rate of excretion in our patients was probably in the neighborhood of the slowest rates of excretion, approximately 0.1 mg. daily, for Aub showed that treatment with cod liver oil and a high calcium diet (used more or less faithfully for all our patients) reduced the rate of excretion to such levels.

It seems probable that the disappearance of the lead line from the bones of growing children is not to be taken as an evidence of complete excretion of lead. It seems more likely that processes of reabsorption and redeposition tend to distribute the lead more evenly in the bone and thus destroy the roentgenologic evidence. For instance, 1 child (case 11) four years after the cessation of pica no longer had a lead line in her bones by roentgen examination but still showed stippled cells in her blood and new neurologic signs of damage to her nervous system. Another child (case 17) three years after her acute episode of lead

poisoning no longer had roentgen evidence of lead in her bones but in connection with a bout of vomiting again showed stippled cells.

Indeed, Aub and his collaborators [2] in discussing the storage of lead in the bones, where they felt it to be relatively harmless, stated: "This desirable situation may not be permanent, for slight changes toward either the alkaline or acid side of the usual hydrogen ion concentration of the organism can readily reduce the stability of the tertiary phosphate." Thus, under chemical shifts common in childhood concentrations of lead known to be significant may be recurrently liberated into the circulation.

It is evident, therefore, that the criteria permitting a diagnosis of lead poisoning are ephemeral in relation to the total process of ingestion of lead and its subsequent excretion from the organism. They depend on the presence in the body fluids or tissues of a certain concentration of lead, and the question of whether the conditions to be discussed are due to the continual circulation of subclinical concentrations of lead derived from the stores in the bones, or to the increase in solubility suggested by Aub [2] or to injury sustained by the developing nervous system at the time of acute episodes must remain a matter of speculation for the present. Possibly all three factors contribute.

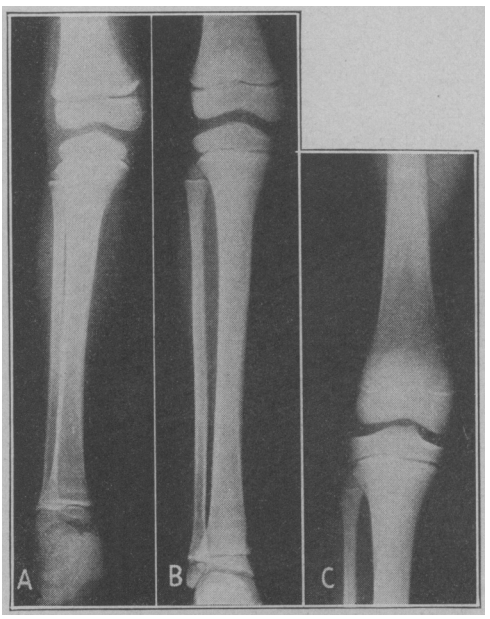

Fig. 4 (case 11).—*A*, at 5 years of age, showing dense bands at the growing ends of the shafts of the long bones; *B*, at 7 years, showing nearly complete disappearance of the roentgen evidence of lead in the long bones; *C*, at 11 years, showing no roentgen evidence of lead in the long bones. At the time roentgenogram *C* was made, the patient's blood showed anemia and stippled cells and she seemed to have a mild encephalopathy.

It is also to be noted that in most instances by the time the difficulties here presented became evident, the diagnosis of lead poisoning, unless established earlier, could no longer be made with certainty by methods now in use. A recent publication by Fairhall and Keenan,[7] describing a relatively simple method for the quantitative determination of lead in urine, may render possible the diagnosis of lead poisoning during the long period of excretion of stored lead after the symp-

7. Fairhall, L., and Keenan, R. G.: J. Am. Chem. Soc. **63**:3076, 1941.

toms and signs of the more acute phases of poisoning have subsided. The usefulness of this method has not yet been proved by wide application.

During the past several years, a number of children who were admitted to the hospital on account of lead poisoning have been repeatedly examined psychologically

Summary Data for 20

Case No.	Age on Admission, Yr.	Source of Lead	Duration of Exposure, Mo.	Pallor	Anemia	Constipation	Peripheral Paralysis	Stippling of Red Cells	Lead Line on Gums	Roentgen Line on Bones	Emaciation	Anorexia	Vomiting	Abdominal Pain	Loss of Strength	Headache	Irritability
1	2 4/12	Chewing paint	..	+	+	..	..	..	..	+	..	+	..	..	..	..	..
2	1 6/12	.....	..	+	+	..	..	+	..	+	..	..	+	..	..	..	..
3	1 6/12	Chewing paint off crib and window sills	12 to 18	+	+	..	..	..	..	+	..	..	..	+	..	..	..
4	2 6/12	Chewing paint off crib, window sills and furniture	20	..	..	..	..	..	..	±	..	+	+	+	..	?	..
5	1 5/12	Chewing paint off crib	..	..	+	..	..	+	..	±	..	..	+	..	+	..	..
6	6 9/12	Chewing paint off crib and furniture	About 5½ yr.	+	+	..	..	+	+	+	..	..	+	+	..	..	..
7	10/12	Chewing paint off crib	Weeks	..	..	..	..	..	..	+	..	+	..	..	..	..	+
8	1 7/12	Chewing paint off crib and window sills	4	..	..	..	..	..	..	+	+	+	..	..	+	..	..
9	3 1/12	Chewing paint off crib and window sills	18	+	+	..	..	..	..	±	..	..	..	..	..	..	+
10	2 6/12	Chewing paint off crib, window sills and furniture	18	..	..	+	+	+	..	+	..	..	..	..	+	..	..
11	2 5/12	Chewing paint off wooden furniture	..	+	+	..	+	+	..	+	+	+	+	+	..	..	..
12	4	Pica (articles not remembered)	..	+	+	..	+	+	..	+	+	+	+	+	+	+	..
13	1 6/12	Chewing paint off crib and window sills	10	..	+	..	..	+	..	+	..	+	+	..	..	..	..
14	11/12	Chewing paint off repainted crib and porch railing	16	..	..	..	..	+	..	+	+	..	..	+	+	..	+
15	3 4/12	Chewing paint	24	+	+	+	..	..	..	+	+	..	..	..	..	..	..
16	4 7/12	Chewing paint off window sills and furniture	24	+	+	+	..	..	..	±	..	+	..	..	..	..	..
17	111/12	Chewing paint	..	..	..	..	..	+	..	±	..	..	+	..	..	..	..
18	10/12	Lead nipple shields used by nursing mother	10	+	+	..	+	+	..	+	..	..	+	+	..	..	..
19	2	Lead water pipes; played with paint	..	+	+	..	+	..	..	+	..	+	..	..	..	..	..
20	3	Chewed paint off crib and window sills	48	+	+	..	..	+	+	+	+	..	+	+	+	+	+

by one of us (E. E. L.) and certain deviations noted. In order to make a systematic study of the matter, the records of the Children's Hospital for the past ten years have been reviewed and 128 cases discovered. The yearly rate of admission has tended to decline from 21 in 1931 to 3 in 1939; such cycles may, however, tend to occur in relation to the interest of the staff in the subject. During

the past year medical records of 71 children (38 boys and 33 girls) living near enough to Boston to be available have been reviewed. Of this group, 12 (9 boys and 3 girls) showed gross evidence of cerebral damage at the time of discharge from the hospital and were not further considered. Of the remaining 59 children

Cases of Lead Poisoning

Encephalopathy	Spinal Fluid	Age at Latest Examination, Yr.	I. Q.	Present Status
.....	.....	9	87	Still reading in a primer; sensorimotor defect
.....	.....	9 2/12	67	Repeated first grade; now doing poor work in second grade
.....	.....	8	82	Unreliable, impulsive behavior prevents friendship; unable to learn in school; cannot write name in spite of two years in first grade; sensorimotor defect
.....	.....	6 6/12	94	Cruel, unreliable, impulsive behavior; runaway; unable to get on with other children or adults; excluded from school because of behavior
.....	.....	6 10/12	100	Not progressing in reading or writing; sensorimotor defects
.....	Total protein, 36 mg.	6 9/12 12 6/12	100 82	A nervous person, doing passing work in seventh grade; no significant variation in sensorimotor sphere
.....	.....	9	103	Serious behavior problem; unable to do school work; referred to remedial class; short attention span; special sensorimotor tests not given
.....	.....	6 10/12	96	After a year in first grade cannot write his name, though he is considered smart; sensorimotor defects
.....	.....	6 4/12	87	After a year in first grade has not learned to write or print his name or recognize any figure; sensorimotor defect
.....	.....	9	105	Lively knee jerks, ankle clonus on left; restless and inattentive at school; is having a hard time; beginning fourth grade; mild sensorimotor defect
.....	Normal	10 9/12 15	92	Babinski sign on right; repeated two grades; had emotional difficulties which were helped by tolerance; difficulties in sensorimotor sphere
.....	Normal	10 6/12	75	Repeating fourth grade; to be put into special class
Choked disks	Total protein, 124 mg.	6	90	Expelled from school for setting fires; sensorimotor defect
Hyperactive knee jerks	Normal	6 1/12	109	Behavior unpredictable and impulsive; unable to learn first grade subjects; death at 6 years and 2 months from agranulocytosis after use of sulfanilamide
Confusion; chorea; tremor	Total protein, 63 mg.; 18 white blood cells	7 7/12	91	Convulsions recurrent at long intervals; unable to learn to read; sensorimotor defect
Convulsions at 4 to 4½ years	.....	7 2/12 12 4/12	90 69	Irregular mental development with extreme unpredictability in results of school work and mental tests; scattered convulsions; enlarged ventricles
Two convulsions in few days before admission	.....	6	94	Convulsions recurrent after 5 years of age; after one year in first grade unable to write name or make figures; irregular mental development; mild sensorimotor defect
Bulging fontanel; edema of optic disks	Total protein, 160 mg.; xanthochromia	7	103	Doing well in school; reads and writes normally for age; sensorimotor tests average for age
Tendon reflexes hyperactive	.....	12	Mental tests not given	Repeated first grade; private teacher at home for one year; then second grade; repeating grades so that he will start fifth grade next fall; enuresis
Hyperactive reflexes; bilateral Babinski sign	Total protein, 108 mg.; white blood cells	9 6/12	79	General intelligence level between 7 and 8 years; doing badly in third grade

discharged as cured, many have received repeated examination by the psychologist, and of these, 20 have now entered the school system. These 20 form the basis of the present report.

A tabular review of the 20 cases is presented in the table. In all but case 2 an adequate source of the lead ingested was disclosed by the history, for this child

lead poisoning was considered an unimportant subsidiary diagnosis in relation to the acute mastoiditis for which he entered the hospital. In 18 cases the lead was obtained by chewing paint off cribs, window sills or furniture. In many instances the parents stated that they had repainted the crib, a practice which would vitiate the use of lead-free paint by the most conscientious of furniture manufacturers. The frequency with which children chewed window sills and painted furniture points to the importance of preventing the use of lead-containing paint on any and all interior finish, on porch railings and on other objects which are in reach of children's teeth. In 1 instance ingestion of lead appeared to be the result of the use of lead nipple shields by a nursing mother.

From the diagnostic point of view there seemed ample evidence to accept these children as having had lead poisoning. All but 1 generously fulfilled the diagnostic criteria outlined in Aub's article, and, in addition, all had roentgen evidence either consistent with (designated by ± in the table) or characteristic of lead poisoning (designated by + in the table). The 1 child who did not have conclusive evidence of lead poisoning was a girl of 10 months who was admitted to the hospital because of anorexia and failure to gain weight satisfactorily since birth. Her diet had been irregularly unsatisfactory throughout her life. Soon after she cut two teeth (all she had at the time of admission), she began to gnaw the paint off her crib. Two days before admission she became irritable. On admission she was a small, irritable baby, continually whining and scratching her palms, soles and genitals. Except for a few beats of clonus at her left ankle, her physical examination was not remarkable. Cytologic examination of her blood showed no pathologic changes, but roentgenograms of her long bones showed dense metallic shadows at the ends of the shafts of the long bones, considered diagnostic of lead. Treatment by protection from ingestion of paint and use of an adequate diet for her age supplemented with dibasic sodium phosphate and cod liver oil was supervised for two months in the outpatient department. The itching of the palms and soles continued. While it may be argued that actual poisoning by lead had not occurred at this time, it was clear that she had ingested paint and stored a heavy metal in her bones in the pattern characteristic for lead.

With respect to the nervous system the cases fell into three groups. In the first group, 9 cases, there was no evidence suggesting involvement of the nervous system. In the second group, 3 cases, there was peripheral neuritis but no clinical sign of cerebral involvement, and in the last group, 8 cases, there was more or less conclusive evidence of encephalopathy. The symptoms and signs among the latter were choked disks, bulging fontanels, hyperactive tendon reflexes, convulsions, confusion, choreiform movements and changes in the spinal fluid. In no case were these symptoms severe, and in every instance they appeared to have cleared completely at the time of the patient's release from the hospital ward. The presence or absence of evidence of involvement of the nervous system bore no relation to the eventual intellectual development of the children; indeed, the only truly successful member of the group had the most severe encephalopathy encountered among the 20 children.

Considering the early age at which the ingestion of lead began, it was difficult to forecast what the intellectual development of any of the children would have been. When the 20 patients were considered as a group, its motor development, as obtained by history, fell in the normal range. Since most of the individuals began to be exposed to lead during middle infancy, other developmental criteria were not applicable. Detailed developmental histories were not available in all instances, but 8 of the children were reported to have sat alone at an average age of 6.9 months, 14 to have walked at an average age of 14.3 months and 11 to have

been using words at an average age of 18 months. These developmental items applied to 15 of the children; 2 more were 10 months old when admitted to the hospital and must have been active to have obtained their lead; for another child the statement of normal development was not documented; for the nineteenth no developmental data were given, and the twentieth child was first seen when she was 6 years old because of failure to do school work as well as had been expected by her family. It was the opinion of the medical staff that these were "normal children" at the time they were originally seen in the hospital. It was obvious that 1 or 2 unsatisfactory children might be found in any group of "normal children" picked in this way at such an early age, but, on the whole, the expectation of reasonable average progress in school was justifiable for the group. The developmental data also disposed of the suggestion that these infants exhibited pica because of mental defect.

After recovery from their lead poisoning these 20 children made an extremely poor record in competition with their fellows. Their difficulties were in relation to both the intellectual and the emotional spheres. The intellectual difficulties were capable of rather exact analysis, and, since in part the emotional difficulties were dependent on them, they will be discussed first.

With one definite and a second possible exception, none of the 20 children succeeded in school. The causes of their failures were probably manifold. For the group as a whole, at about the time of entrance to school the intellectual ratings averaged 90, with a spread from 67 to 109. The usual correlation between the intelligence quotient and the ability to learn in school did not hold for the group, the second most successful child having an intelligence quotient of 82 while several with quotients above average were unable to do school work. For the 5 children with intelligence quotients ranging from 67 to 85 slow progress in school seemed well enough explained by the low intelligence level. These children did not show the mental irregularities to be discussed in relation to the children with better intelligence levels, since special defects merged into the general intellectual inadequacy.

Throughout the psychologic examination of the children with good intelligence levels and poor scholastic attainment, sensorimotor defects were found in most instances. This is not the place to discuss the tests used to show such defect, since they must be varied to suit the age of the child examined, since they must be given by an experienced psychologist trained in their use, and since they have been well described in the literature of psychology. In brief. however, in the earlier age groups sensorimotor defect was shown by inability to manipulate blocks, to fit forms into holes in a form board and to perform similar acts. In the preschool years inability to copy simple figures, such as crosses, triangles and squares, proved to be important. In the school ages inability to reproduce by memory the designs of the Ellis Visual Designs Test [8] or of the Pintner-Cunningham Test No. 7,[9] to deal with the block designs of the Wechsler-Bellvue Test [10] or to build up the associations to pictures necessary for success in the Wood Picture Completion Test [11] were found. All of these tests are designed to bring out a subject's ability

8. (*a*) Bronner, A., and others: A Manual of Individual Mental Tests and Testing, Boston, Little, Brown & Company, 1927. (*b*) Wood, L., and Shulman, E.: J. Educ. Psychol. **31**:591, 1940.

9. Pintner, R., and Cunningham, B. V.: Primary Mental Test, New York, World Book Co., 1922.

10. Wechsler, D.: The Measurement of Adult Intelligence, Baltimore, Williams & Wilkins Company, 1939.

11. Wood, L.: J. Genet. Psychol. **56**:383, 1940.

to deal with shape, direction, space and projected imagery, all matters of the utmost importance in a child's success or failure in the fundamental school of technics. In the grades, inability to learn to write or read or deal with numbers in arithmetic was the result of such defect, even though verbal ability might be good. By contrast, many of the children showed normal or even superior abilities in the language field, and on this account several of the most severely handicapped children made relatively high scores on the Stanford-Binet tests. Such children were frequently socially responsive, especially in their preschool years, and were considered "bright" by parents and teachers, the latter often stating that the children could do better if only they would try.

One child, as previously intimated, appeared to have recovered completely; at 7 years his intelligence quotient was 103 and he was progressing satisfactorily in school. At 25 months, however, he presented intellectual difficulties of a sort that were permanent in other children of the group. He was an attractive, good-natured boy, socially responsive, pleasant, imitative and amenable. His understanding of language allowed him to carry out simple commands in the test situations appropriate for a 21 month old child. He associated the proper pictures with Mother Goose Rhymes, and he liked to listen to verses. In the use of language he was more like an 18 month baby in that he had a vocabulary of only half a dozen words and did not combine words. From the point of view of adaptive behavior, he pushed single blocks imitatively but made no effort to aline them. He piled large blocks at home but could not make the necessary adjustments to pile small cubes, being less adequate than most 18 month old babies in this respect. He adapted the circle to the form board, but though he placed the other forms over their holes he could not make the adjustments necessary to insert them, again dealing with shape and spatial orientation like an 18 month baby. With a crayon he scribbled and made strokes as do most babies of 21 months. In summary, he was imitative and resourceful, but the sensorimotor element in the adjustment of blocks and the fitting of forms into a board was definitely not as advanced as his understanding of language and his social response. As he grew older, these sensorimotor disabilities diminished, and at 7 years he appeared to be intact. His ability as a baby to scribble imitatively with a crayon was not found in any of the other children of the group who were examined early. He was the only child in the entire group who did well in school.

One other child in the group kept up with her class in school with great difficulty. She was a pleasant, docile person, recognized as relatively incompetent by her family. Her intelligence quotient at 7 years was 102, but at 12 years it was only 82. Sensorimotor capacity as shown by the Ellis Memory Test was below the twenty-fifth percentile for her age, and her ability to organize her visual impressions as measured by the Wood Picture Completion Test was in the second decile for her age. Thus her difficulties were most obvious in the sensorimotor sphere, but they were not far out of line as compared with her general intellectual level.

Eleven children were examined when they were between 18 months and 2½ years of age. Each of the 11 exhibited more advanced reactions to language tests than to the tests of sensorimotor control. Seven were using words as well as one would expect for their age, whereas in 4 speech was delayed, but the play with blocks and forms was consistently less adequate than speech. In all but the child already discussed as completely recovered a definite lag in the ability to make imitative adjustments with the crayon was found.

Six children were examined between the ages of 4 and 5 years. They answered comprehension questions well; some counted, and some even named colors. In

each instance there was more than the average difficulty in adjusting blocks in picture puzzles and in reproducing drawings. Two of the children, approaching 5 years, had begun to be interested in making letters, but their copy of letters was disoriented and they were unaware of the displacement. One particularly attractive little girl aged 4 7/12 years made an extremely favorable impression in the ward. She was so friendly and cooperative with the doctors that one of them said, "I should like to adopt that child." On the Stanford-Binet scale she had a mental age of 4 8/12 years and an intelligence quotient of 112. The comprehension questions were answered adequately not only on the Stanford test but on several other tests. Although she matched outline shapes, showing adequate visual perception, she had more difficulty in fitting the blocks in the Seguin form board than most 3 year old children. She had more than average difficulty in block building, and her hand was unsteady as she traced the Porteus Cross. Although her ideation was good in the drawing of a man and she made a figure with head, eyes, legs and arms, the whole figure was disoriented. Here again are found good ideation, verbalization and social response for a 4 year old child but less competence than that of the average 3 year old child in the adjustment of forms.

These deficits in the younger children did not in themselves seem more than minor deviations; but when children who still exhibited them encountered the school system, severe difficulties in learning became evident. Two of the children have been promoted each year, to the fourth and seventh grades at 9 and 12 years respectively, but complaints of inattention, restlessness and inaccurate work continue to be made about them. Of the children in the first grade, the comment has been made that they "seem smart enough but do not learn." Another child is "bright but restless and does not concentrate, is learning a few words with help from his mother at home but is having a terrible time with writing." A mother reported: "He is smart, and I have no complaints. Although the youngest boy in his group, he is going to make his first communion with the others." When this situation was explored a little more fully, it was found by the psychologist that though he was 6½ years old and had an intelligence rating of 96, at the end of the first year in school he did not recognize a single word and had not learned to write his name. He could make numbers, but they were disorientated. In general, these comments reflect the frustration of parents and teachers with these responsive, verbally able children who because of sensorimotor defect could not learn the technic taught in the first grades in school. The table summarizes the school situation in regard to all 20 children, and their clinical histories appended afford more detail.

In addition to difficulties in the general intellectual and sensorimotor spheres, other evidences of interference with the normal development of the nervous system were found scattered through the series. For a few children successive psychologic examinations showed significant drops in the intelligence quotient. These drops were felt to be the result of failure of mental development rather than of actual mental deterioration. As a result, though the chronologic age advanced, mental growth did not keep up with it. Cases 4 and 11 are examples. In case 16 the fall in intelligence quotient was associated with recurrent convulsions and such great variability in intellectual function from day to day that it seemed likely that mental deterioration had actually occurred.

Recurrent convulsions appeared in 3 of the children, at 4, 4½ and 5½ years of age. Of these, 1 (case 16) showed enlarged ventricles on pneumoencephalographic examination at 12 years of age. One girl who had peripheral neuritis as a baby acquired a positive Babinski sign, first noted when she was 11 years old, and 1 boy, who was discharged from the hospital as well at about 3

years of age, had hyperactive reflexes and sustained clonus at the left ankle when reexamined at 9 years.

Behavior difficulties were common throughout the series. Much of this behavior could be classified as "forced reaction to stimuli in the environment" described by Strauss and Werner [12] as an evidence of cortical damage. It was apparently the result of loss of the normal inhibitory function, thought to reside in the cortex. It was usually described as unreliable impulsive behavior, cruel impulsive behavior, short attention span and the like. It made several of the children friendless and difficult at home and in school. Three were excluded from school on the basis of behavior, 1 for setting fires in the school, another for repeatedly getting up and dancing on the desks and other furniture and the third for sticking a fork into another child's face.

In some children the untoward behavior seemed to occur under any and all circumstances. An example is a boy (case 14) who first came to our attention at 23 months because of irritability. His lead poisoning was diagnosed and treated, and he was sent home as cured. From that time on, he presented a severe behavior problem, with crying, irritability, refusal to eat, soiling, wetting and abdominal pain. His mother, a good-natured, kindly, humorous woman who handled him with admirable tolerance and insight, was given many brisk talks by the medical staff on how children should be brought up. From time to time he was admitted to the hospital in an attempt to modify his behavior or because of a suspicion of appendicitis. In the wards, his behavior, though not as flagrant as at home, continued essentially unchanged. It was felt that his improvement in the wards was due largely to the fact that a routine requiring much of one person's time could be established for him and in addition to the fact that the nurses and doctors caring for him had no deep emotional attachment to him. Fundamentally he remained irritable, impulsive and distractable, and it was impossible for teachers to deal satisfactorily with him in the group situations in the kindergarten and the first grade.

On the other hand, some of the children acquired behavior difficulties motivated in part, at least, by the frustration resulting from the impact of scholastic disciplines on their intellectual deficits. An example is a girl (case 11) who was first thoroughly studied at 5 years of age because of peripheral neuritis due to lead; at that time no psychologic difficulties were suspected. At 11 years of age she returned to the hospital because of difficulty with school work and emotional disturbances. She was sullen, withdrawn and insecure and on medical examination was found to have positive Babinski signs (a new observation) and stippled cells in her blood. Psychometric examination showed sensorimotor defects, and with the assistance of an extremely intelligent school supervisor, allowances for her disabilities were made and treatment for lead poisoning instituted. The behavior difficulties smoothed out at once, and when last seen, at $15\frac{4}{12}$ years, she was a pleasant, attractive-looking girl, with a quiet and poised manner, getting along fairly well in the eighth grade but unable to do arithmetic. She had repeated two grades, and her family were hoping mildly that she might reach high school. She had made real gains in the specific sensorimotor field, her score of the Ellis Memory Test being normal for her age and her performance with the Wechsler-Bellevue Block Designs and the Wood Picture Completion Test more nearly up to expectation for her age than before. These findings suggested that her psychologic state at the time of her depression was due, in part at least, to a reactivation of her latent lead poisoning.

12. Strauss, A. A., and Werner, H.: Am. J. Psychiat. **97**:1194, 1941.

The conditions imposed by present day culture on children with good intelligence quotients and sensorimotor defects are very difficult. Their excellent verbal capacity and social responsiveness make it seem impossible to parents and teachers that they could not learn if only they would. A good example is presented in case 15. At 8 years the patient was a pretty, outgoing child, large for her age, with an intelligence quotient of 91 and no ability to learn to read or write because of sensorimotor defect. The choices before those responsible for her school planning were three. The first was to keep her back in the first grade, where her large size and advanced age combined with her inability to compete in learning would certainly humiliate her. The second possibility was to put her into the second grade, where the defective preparation and learning defects would be certain to frustrate her. The last possibility was to let her go nominally into the second grade but to protect her from competition with the other children by giving her tutoring in the academic subjects. Such a plan could be worked out only in a tolerant and well staffed public school system.

Lead poisoning in these 20 children began in infancy. In order to obtain the lead, all but 1 must have developed at an average rate from the motor point of view. None of them exhibited evidence of severe acute encephalopathy, yet only 1 lived up to the promise of his early development. In some, such failure was parallel to a generalized defect in mental development, resulting in a readily recognizable lowering of the intelligence quotient. In others, though the intelligence quotient remained well within normal limits, a specific failure of development in the sensorimotor sphere was the outstanding finding.

That failure of intellectual development is somehow connected with improper development of the cerebral cortex is well accepted. Sensorimotor defect has been studied by several observers. Goldstein and Scheerer [13] found it especially in relation to abstract designs, as an evidence of cortical disease or injury. Wechsler [10] found that failure in his block design tests correlated well with organic cerebral lesions, and Lord and Wood [14] have shown that failure in the sensorimotor sphere was common in a group of children with known cerebral disease and uncommon in a group of children under treatment for psychiatric disturbances.

There is ample evidence that the cortex of an infant is not as yet completely active functionally. From the motor standpoint, Kennard [15] showed that decortication of infant monkeys produced little disability as compared with decortication of adults, and Byers [16] showed that hemiplegia in early infancy produced a distinctly different clinical picture from that found after the upright posture had been attained. The normal processes of intellectual growth and development are thought to be due to processes of maturation taking place in the cortex. It seems likely, therefore, that the lead in the circulation of an infant in some way interferes with the changes normally occurring in the cortex and in a high percentage of cases prevents the normal growth and development of the cortex.

SUMMARY

A follow-up study of 20 school children who had been hospitalized in infancy or early childhood because of lead poisoning has been presented. None of them

13. Goldstein, K., and Scheerer, M.: Abstract and Concrete Behavior, Evanston, Ill., American Psychological Association, Inc., 1941, vol. 53, no. 2, p. 131.

14. Lord, E. E., and Wood, L.: Am. J. Orthopsychiat. **12**:414, 1942.

15. Kennard, M. A.: Relation of Age to Motor Impairment in Man and in Subhuman Primates, Arch. Neurol. & Psychiat. **44**:377 (Aug.) 1940.

16. Byers, R. K.: Evolution of Hemiplegias in Infancy, Am. J. Dis. Child. **61**:915 (May) 1941.

exhibited striking evidence of encephalopathy during their primary admission, and all were adjudged to have made a complete recovery from lead poisoning when discharged from the original hospitalization.

The length of the cycle of ingestion, storage and elimination of lead in relation to the relatively short duration of the symptoms and signs allowing a diagnosis of lead poisoning is emphasized. It seems probable that lead poisoning of the sort here discussed can at present be recognized in only a small percentage of cases.

Failure of the normal processes of growth and development of the cortex prevented all but 1 of the 20 children from progressing satisfactorily in school.

Lead poisoning is a serious disease developing from entirely man-made hazards, which should be controlled by appropriate legislation.

REPORT OF CASES

CASE 1.—A boy of 2 years 4 months was admitted to the outpatient department because of lack of appetite. He was the only child of healthy parents. He sat at 7 months and walked at 14 months but was talking only a little at the time of his first visit. He had chewed the paint "off everything" when he was smaller, though at just what age and for how long was not stated. He was completely out of control of his parents at the time of his admission, screaming for what he wanted, refusing to give up his bottle and exhibiting impulsive and irritable behavior. He had been "pale for a long time."

Physical examination showed the boy to be small and pale but otherwise revealed nothing remarkable. A blood smear showed moderate achromia but no stippled cells. His urine was normal. Roentgenograms of his long bones showed dense bands of metallic deposit at the growing ends of the shafts, characteristic of lead poisoning. Twenty drops of halibut liver oil and a high milk diet were prescribed, and later iron and ammonium citrates was added.

After visiting the hospital he continued to chew the paint off the window sills and furniture at home, but finally this habit stopped spontaneously. At 4 years he was still small but had become shy and repressed. Psychologic appraisal showed poor speech and considerable difficulty in handling test materials, suggesting a general mental level below average, with mental irregularity due to specialized difficulties.

At about this time his mother died of pneumonia and he was boarded out. Treatment with vitamin D in various forms and dicalcium phosphate was, however, carried on. He continued to be rather unsatisfactory physically, intellectually and emotionally.

At 9 8/12 years he was in the third grade in school, though he had failed to learn to read. At home he was difficult and impulsive. A psychologic appraisal indicated an intelligence quotient of 87, with special disabilities in the sensorimotor sphere. His score on the Wood picture completion test was at the third decile for his age, and on the Ellis visual design test it was worse than any calibrated score.

CASE 2.—A boy of 1½ years was admitted to the hospital because of projectile vomiting of ten days' duration. He was born normally at full term and did well as a baby. His dentition was late, beginning at 14 months, and he began to walk and talk just prior to his entry to the hospital. Two months before his admission scarlet fever and running ears developed, one of which continued to discharge to the time of admission. Two weeks before entry he began to vomit projectilely at night, and he continued to do so to the time of admission.

Physical examination showed a pale child with a discharging right ear. Roentgenograms showed a clouded and eroded right mastoid and a broad band of metallic deposit at the growing end of the long bones, characteristic of lead poisoning. A roentgenogram of the abdomen showed much dense flocculent material, probably paint, in the intestinal tract. His blood showed a secondary anemia (hemoglobin content 55 per cent and 4,100,000 red cells), many stippled cells on smear and an elevated percentage of polymorphonuclear leukocytes.

A mastoidectomy was performed on the right side, and treatment with calcium gluconate, dibasic sodium phosphate and viosterol in oil [17] was carried out. He did well and was discharged after three weeks in the hospital. At this time psychologic appraisal showed that he handled materials like a 15 month old baby, with no special defects.

At 9 2/12 years psychologic appraisal showed an intelligence quotient of 67, with no specialized defects. In school he was in the second grade, having repeated the first grade.

17. The preparation used in this and succeeding cases contained 10,000 U. S. P. units of vitamin D per gram.

CASE 3.—A boy of 18 months was admitted to the outpatient department because of irritability and crying at night as if in pain. His development was normal. He sat up at 7 months, walked at 13 months and was using words at 18 months. When about 1 year of age, he began to chew the paint off his crib and had continued to do so. In spite of this, he was sent home as presenting a behavior problem.

Eight months later he reappeared with the same complaints and vomiting. His crib had been covered with cloth, but he had taken to chewing the paint off window sills. Roentgenograms of his long bones showed a narrow band of metallic deposit at the growing ends of the long bones, but in spite of letters to his parents he did not report for treatment for six more weeks. At this time his hemoglobin content was 40 to 50 per cent and a blood smear showed no stippled cells. Cod liver oil (½ ounce [15 cc.] daily) and dibasic sodium phosphate were prescribed, and a further warning about ingestion of lead was given to the parents. A month later roentgenograms showed much more pronounced metallic deposits at the growing ends of the shafts of the long bones.

A few weeks later vomiting recurred once or twice daily, and the boy became irritable. Though he had been prevented from chewing paint, it was discovered at this time that the pipes in his home were leaden. He still had a secondary anemia (hemoglobin content 59 per cent and 4,500,000 red blood cells), but on several examinations no stippled cells were found. At this admission, when he was 2½ years old, he was seen by the psychologist, who found his development below the standards for the 2 years, in contrast to his chronologic age. He was discharged from the hospital; treatment with disodium phosphate, viosterol in oil and phenobarbital was continued at home, and the health department was asked to do what was necessary to free the drinking water from lead.

At 8 years and 4 months he had difficulty in getting on with other children because of his unreliable, impulsive behavior. He had not acquired his first grade technics, in spite of two years of instruction, and was not able to write his name. Psychologic appraisal indicated a mental age of 6 11/12 years and an intelligence quotient of 82.

CASE 4.—A girl of 2½ years was admitted to the hospital because of temper tantrums and abdominal pain occurring during the nine months prior to admission. Her development had been average. She sat at 6 months, stood at 8 months and talked at 15 months. From the time she was 9 months old she exhibited marked pica, and until the time of admission she continued to chew paint from her crib, the furniture and window sills. At about 20 months she began to have attacks of screaming and banging her head, which lasted fifteen to ninety minutes, made her blue and tremulous and left her exhausted. During the two weeks before her admission she had complained of abdominal pain and vomited projectilely several times. Although she began to use single words at 15 months, she was using only single, rather unintelligible words at the time of admission.

Results of the physical examination and laboratory investigation were not remarkable. Roentgenograms of the long bones showed dense bands along the diaphysial margins, consistent with lead poisoning.

In the hospital she fell out of bed and was taken home on this account by the parents. Four years later, at the age of 6½ years, she returned, having been excluded from school because she danced on the furniture in the classroom and because of similar uncontrollable behavior. In addition, she was cruel to animals, bit people for the pleasure of seeing them bleed and ran away by hopping street cars. In the interim her father and mother had separated several times and divorce proceedings had been instituted. She was unable to get on with other children because she always insisted on having her own way. A large variety of punishments had failed to control her.

Physical and laboratory investigation showed nothing unusual.

Psychologic investigation showed an intelligence quotient of 95 (Terman L), but a marked lack of mental control. She could not apply herself to any problem for more than a very short period, and she was unable to learn in school situations, even with individual instruction. Her auditory memory span was extremely short. She could repeat only three digits correctly. She could not learn a two line nursery rhyme. She could not learn the names of the individual letters comprising her name, though she could print her name. She was excluded from school because she danced about on the desks and piano.

CASE 5.—A boy of 17 months was admitted to the hospital because of diarrhea and vomiting of three days' duration. He had been born normally and had developed normally, sitting up at 6 months and walking at 13 months. At the time of his admission speech had not developed. He had had frequent bouts of diarrhea and vomiting, usually accompanying infections of the upper respiratory tract, which had begun when he was 2 months old. For some months before admission he was known to be chewing the paint off his crib, which was made of metal.

On admission to the hospital he showed mild dehydration and was drowsy. Except for slight evidences of rickets, the physical examination showed no abnormality. Two days later blood smears showed a few stippled cells. Roentgenograms of his long bones at this time showed a narrow band of increased density at the ends of the diaphyses, consistent with lead poisoning.

He was treated for dehydration and was sent home on an average diet with added viosterol in oil (15 drops), dibasic sodium phosphate (30 grains [0.19 Gm.]) daily and calcium lactate (30 grains [0.19 Gm.]) daily.

He returned a year later with the same complaints, and though he was 2½ years old he had not acquired speech. In spite of his mother's efforts, he had continued to chew paint.

Physical examination showed a screaming, unresponsive child who continued to bang his head about and beat it with his hands for some time after admission. His gait seemed definitely unsteady, but otherwise nothing remarkable was found.

His blood showed a mild secondary anemia, and on spectroscopic examination many times the normal amount of lead was found in the blood. After three weeks of treatment with cod liver oil (2 drachms [7.75 cc.] daily) and a high calcium diet, this level was found to be greatly diminished. Roentgenograms of his long bones still showed heavy bands at the end of the shafts.

His irritability diminished, but psychologic examination showed that he did not use words and suggested sensorimotor disturbances.

A few weeks later he returned with an acute otitis media and a digestive upset, which subsided quickly. At this time his blood serum showed a trace of lead on spectroscopic examination, while the clot showed a good deal more. It was reported that his pica had disappeared.

At 3 years and 8 months he returned, drowsy and dehydrated after several days of projectile vomiting. His fundi were hyperemic, and the optic disks were poorly outlined. Otherwise the results of the examination were unremarkable. A lumbar puncture was not done, but the other usual laboratory procedures indicated no abnormal conditions. Roentgenograms of his bones no longer showed any suggestion of deposits of lead.

At 4 years and 5 months he was readmitted, with pneumonia, from which he recovered without incident. After this he was transferred to the neurologic ward for appraisal. His language development was normal for his age, but he had difficulty in the use of crayons and adjustment of forms and blocks, diagnostic of sensorimotor impairment. He has returned for psychologic check-ups from time to time because has has been unsatisfactory to his teachers. At 6 11/12 years his mental age (Stanford-Binet) was 6 11/12 years and his intelligence quotient was 100, but it was impossible for him to do any of the cube designs which are usually satisfactorily dealt with at 6 years. He was not progressing in reading or writing in school.

CASE 6.—A girl of 6 years and 9 months was admitted to the hospital because of vomiting of six days' duration. Her own past history and that of the family were irrelevant except that she had chewed the paint off her crib and had exhibited other forms of pica from infancy until about six months before admission to the hospital. She had entered school in the first grade eight months before, and disappointment was expressed because of her failure to learn as well as had been expected. There had been one previous bout of vomiting of two days' duration initiated by a blow on the abdomen six months before admission. Abdominal pain and vomiting three or four times in twenty-four hours had begun six days before admission and were tending to become more severe.

Physical examination showed a thin, pale, dehydrated girl with teeth in poor condition and a pronounced lead line on the gums. Her fundi showed no abnormalities, and no abnormalities were found on neurologic examination. Her blood showed a secondary anemia (hemoglobin content of 50 per cent and 2,600,000 red cells), and about 2 per cent of the red cells were stippled. Spectroscopic examination of her blood and spinal fluid showed a large and definitely pathologic amount of lead present. The total protein content of the spinal fluid was 36 mg. per hundred cubic centimeters. Otherwise the fluid was unremarkable. Roentgenograms of the long bones showed some density at the growing ends of the shafts of the long bones insufficiently marked to allow a definite roentgen diagnosis of lead poisoning. A roentgenogram of the abdomen showed much dense flocculent material in the intestines consistent in appearance with flecks of paint.

Treatment with sodium phosphate and milk did not alleviate the vomiting, and because of continuing dehydration and acidosis she was given physiologic solution of sodium chloride subcutaneously and 10 per cent solution of dextrose intravenously. Two hours after this she had a generalized convulsion, which was relieved forty-five minutes after its onset by paraldehyde given rectally. She was then treated with calcium gluconate, milk, sweetened

fruit juices and atropine. She gradually improved and was discharged on the twenty-second hospital day, eating well. At the time of discharge an increase in density of the dense bands at the ends of the shafts of the long bones was noted on roentgen examination.

A psychologic examination given the day before her discharge showed an intelligence quotient of 100 and adequate response to all tests.

Two years later she was seen in the outpatient department because of nervousness and crying in her sleep. Her physical examination and her blood showed no abnormalities, and roentgenograms of her long bones showed faint scars of the previous dense bands in the diaphyses.

At 12½ years she was considered a nervous person by her father, but she was doing passing work in the seventh grade in school. Mental tests revealed an intelligence quotient of 82, with no significant deviations.

CASE 7.—A girl of 10 months was admitted to the hospital because of poor appetite and refusal of food since birth and irritability for two days. She was born normally at full term, and weighed 6 pounds 7 ounces (2,920 Gm.). Since birth she had eaten poorly and gained slowly. Orange juice and cod liver oil had been refused or vomited, apparently quantitively. Since cutting two teeth (time unstated), she had chewed the paint off her crib.

On physical examination she was a small, irritable baby, constantly scratching her palms, soles and genitals. There was ill-sustained ankle clonus on the left, but otherwise results of physical and neurologic examinations were unremarkable. Her blood showed no anemia and no stippling.

Roentgenograms of her long bones, taken because of the suspicion of scurvy, showed a line of increased density at the growing ends of the diaphyses, characteristic of lead poisoning.

She was treated with an average diet, cod liver oil and dibasic sodium phosphate, which she took well, but the scratching of her palms and soles continued in the hospital and for the two months during which she was followed in the outpatient department.

She was seen at long intervals because of infections until she was 6 years old, when the school sent her back to the hospital because she was irritable, nervous and inattentive. At 9 years she returned with the same complaints, to which was added the statement that in spite of an adequate intelligence rating she would not do her school work. She was considered to present a serious behavior problem and had been referred to a remedial reading class. Her attention span was very short. Her intelligence quotient was 103 (Stanford-Binet), but sensorimotor tests were not given.

CASE 8.—A boy of 19 months was first seen at the hospital because of poor appetite and loss of weight. His family history and his past history were not remarkable. At about 14 months he stood up and chewed paint off his crib, and later he chewed it from window sills and other beds.

Although he stood alone at 14 months, he did not walk alone until he was 23 months old, apparently because of loss of strength.

At 2 years and 2 months he was admitted to the hospital because of refusal of food and violent behavior, consisting of tearing up toys, defecating on the breakfast table and the like. Physical and neurologic examinations revealed nothing remarkable. Laboratory examination revealed no abnormalities of the urine, blood or spinal fluid.

Roentgenograms of the long bones showed broad bands of increased density at the ends of the long bones, consistent with lead poisoning.

Psychologic examinations were given at 2 2/12 and 2 7/12 years. On the second examination the following facts were noted: "In the past five months the child shows considerable advance in vocabulary, but there has been little or no advance in his ability to manipulate blocks and forms or to use a crayon imitatively."

At 6 5/12 years he had a mental age of 6 2/12 years (Stanford-Binet) and an intelligence quotient of 96. In spite of this, he had not learned to write his name although he was at the end of the first grade. With four reversals, he made the numbers to ten. He had not learned to read at all. For example, he read "boy, cat, dog, play" as "black, red, gray."

CASE 9.—A boy of 3 1/12 years was admitted to the outpatient department because of pallor and eating paint and dirt. He was the third child of healthy parents, was born at term by low forceps delivery and seemed vigorous at birth. He cut his first teeth at 6 months, walked alone at 14 months and was using words purposefully at 16 months. From the time he stood up, at 10 or 11 months, he chewed the paint off his crib and window sills. He was irritable and cranky.

On physical examination he was pale and difficult to manage. His blood showed a hemoglobin content of 55 per cent; his urine contained a slight trace of albumin, and roentgenograms of his knees showed dense bands at the growing margins of the bones, characteristic of lead

poisoning. He was put on a high calcium diet with added iron and viosterol, and warning about further ingestion of lead was given his parents. With this treatment his family reported that he became much less irritable. His hemoglobin content reached 85 per cent in two months, and he was dismissed as well.

At 6 1/12 years of age he returned in response to a follow-up letter. After a year in first grade he had not learned to write or print his name, to recognize any words or to make figures. He was less adequate with picture puzzles and in copying designs than most 5 year old children, while his verbal capacity was relatively adequate. By the Stanford-Binet standards he had a mental age of 5 1/12 years and an intelligence quotient of 87.

CASE 10.—A boy of 2½ years was admitted to the hospital because of fever and difficulty in walking of a few days' duration. His father, aged 21 years, had suffered for nine years from attacks of syncope. His mother and one younger brother were well. He was born normally and did well as a breast-fed infant. He walked at 1 year, began to talk at about 2 years and was talking well at the time of admission. He had had a few acute infections of the respiratory tract but had otherwise been well. From the time he learned to stand, he had chewed the paint off his crib, window sills and furniture, a habit which had never been controlled. He had always been constipated. Three days before his admission to the hospital a fever developed, and two days before admission he became weak and dragged his legs in walking.

Physical examination showed a rather uncooperative child, whose left knee jerk and both ankle jerks were absent and who showed bilateral foot drop. His blood showed a hemoglobin content of 85 per cent and a red cell count of 5,000,000, but on smear 0.7 per cent of the red cells were stippled. His urine and spinal fluid were normal. Roentgenograms of his legs showed bands of moderately increased density at the growing ends of the diaphyses suggestive of lead poisoning. He remained in the hospital twenty-four days and was treated with daily doses of calcium lactate (30 grains [0.19 Gm.]) and viosterol in oil (10 drops). The weakness cleared up gradually, more quickly on the right side than on the left, and on the twenty-fourth day he was discharged in good condition, having gained 3 pounds (1,307 Gm.). His parents were instructed to guard against further ingestion of lead and to continue the use of viosterol and a high calcium diet.

He was seen irregularly in the orthopedic department during the following year for care of the pronation of his feet. Two years later incisions were made in his ears because of acute otitis, without incident.

At 7½ years he was brought to the hospital because of nervousness. The father and mother had separated, and the children were in the care of their grandmother. The boy was in the third grade and was reported to be doing well. The mother was told to improve her discipline.

At 8 10/12 years he returned with the same complaints. His knee jerks were lively, and there was clonus at the left ankle. The school reported that he was restless and inattentive, especially in the first two grades.

Psychologic examination (Stanford-Binet) showed a mental age of 9 1/12 years and an intelligence quotient of 105, but his rating on the Ellis memory test was below the twenty-fifth percentile for his age. He was beginning the fourth grade.

CASE 11.—A girl of 2 5/12 years was seen in the outpatient department because of vomiting of several weeks' duration and occasional abdominal pain. Except that her general condition was poor and that she was pale, results of her physical examination were not remarkable. Her blood showed a mild secondary anemia and her urine a moderate amount of pus. Although an appointment was made for a cystoscopic examination, she was not seen again until she was 4¼ years old, when she came with the history that the vomiting and abdominal pain had continued intermittently and that at fourteen months and again at two months before her admission to the hospital she had had fever and pains in her extremities. The account suggested muscular rather than articular pains. When she tried to get up after the pain subsided, she could not walk because of weakness and had marked foot drop. When she was seen after the second attack, her knee jerks were present. No other reflexes are recorded. A history of chewing wood was obtained, and her blood was examined for stippled cells, without success.

A year later, at 5½ years, she was admitted to the hospital with the same complaints. Her history revealed no new facts save that her development had been average. She had held up her head at 5 months and walked at 1 year. The age at which speech developed was not stated. She had never been well and strong since the original attack of pain and weakness at the age of 3 years; though partly recovering, she had relapsed each late summer and fall. At this time her family stated definitely that in chewing wood she had chewed painted articles of furniture.

On this occasion she showed pronounced weakness of all her voluntary muscles, with complete paralysis of her diaphragm, demonstrated by fluoroscopic examination. There was no evidence of intracranial pressure. Her tendon reflexes were abolished except for both biceps jerks, which could be obtained. Sensation seemed unimpaired. Her blood showed 1.8 per cent stippled cells and a mild secondary anemia. Roentgenograms of her long bones showed narrow dense bands at the growing ends of the shafts of all the long bones and, in addition, three less well defined areas of density proximal to these suggesting recurrent lead poisoning. Her spinal fluid was not remarkable.

A definite diagnosis of peripheral neuritis due to lead was made, and an attempt was made for two days to cause excretion of the lead with use of ammonium chloride, but she vomited so severely that this was abandoned and treatment with calcium lactate and cod liver oil started, and she was transferred to the neurologic ward, where she was given massage and exercises in a tank. With this treatment her neuritis cleared slowly, and after three months of treatment her blood still showed 2.3 per cent stippled cells.

Two years later, at 7 years of age, she was seen in the outpatient department in response to a follow-up letter and was thought to be entirely well. Roentgenograms at this time still showed faint bands of density some distance behind the growing ends of the long bones, thought to be due to the old deposits of lead.

Four years later, when she was 11, she was returned to the hospital because of difficulty with school work and emotional disturbances, depressional in character. She had been free of physical symptoms in the meantime. Physical examination showed slight pallor, slight weakness of the right side of the face, lively tendon reflexes and persistently positive Babinski signs on both sides. Her blood showed a secondary anemia (hemoglobin content 50 per cent and red cells 4,000,000) and a few stippled cells. Her urine showed a very slight trace of albumin. Spectroscopic examination of her blood, urine and spinal fluid showed about one hundred times the concentration of lead found in normal persons. In spite of these results, roentgenograms of the long bones showed no evidence of lead deposits. Encephalograms showed no gross evidence of cerebral damage. Treatment for lead poisoning was again instituted, and concessions commensurate with her abilities were arranged.

At 15½ years she was a quiet, well poised girl, getting along happily but slowly in the eighth grade, though totally unable to do arithmetic. The family hoped she might get into high school and possibly finish it, but they were no longer pushing her scholastically.

Repeated psychologic examinations were made. At 5 8/12 years her mental age (Stanford-Binet) was 6 2/12 years and her intelligence quotient 106. The test showed irregularities and inability to copy the square at the 4 year level, and her hand was unsteady as she traced the Porteus cross.

Her poor sensorimotor control was further shown by her inability at 6½ years to copy letters or write her name.

At 10 10/12 years her intelligence quotient was 92 (Stanford-Binet), but in the Stanford reading test her paragraph meaning scored at grade 2.8, her auditory memory was poor, and her sensorimeter capacity as tested by the Ellis Visual Designs test was very poor (score 3).

At 15½ years she came in again. The Stanford-Binet test was not repeated, but the sensorimotor tests were given once more. She had made a real gain in this respect. Her score was average for her age on the Ellis Visual Designs test. She succeeded on three of the five block designs, though they were more than usually difficult for her. On the Wood Picture Completion test her score was 113, which was the third decile for her age.

CASE 12.—A 4 year old girl was admitted to the hospital because of anorexia, loss of weight and progressive weakness of eight weeks' duration. She was the fourth child of healthy but divorced parents and did well throughout infancy. She sat up at 6 months, walked at 14 months and talked at 18 months. Pica was noticed by the mother at the beginning of the present illness; the child broke bits of plaster off the wall and ate them. No certain consumption of paint was remembered. Weakness developed rather rapidly over the period of eight weeks so that she could not support her weight or pick up objects.

Examination showed a weak, emaciated little girl with great muscular weakness but no complete paralyses. The diaphragm hardly moved actively at all. All tendon reflexes were absent. There was marked bilateral wrist drop and toe drop. Sensation to a pin and to touch appeared intact. No evidence of intracranial pressure was found.

Laboratory investigation showed a secondary anemia (hemoglobin content 60 per cent and red blood cells 3,200,000) and about 1 stippled cell per high power field on smear. Her spinal fluid was normal. Roentgen examination showed definite bands of metallic deposit at the ends of the shafts of the long bones, considered typical of lead.

The patient remained for two months in the hospital on a high calcium diet with added iron and cod liver oil. Her anemia improved fairly rapidly, but her weakess was still

crippling. She remained in a convalescent home another four months. Two months before her discharge from the home she was walking, and at the time of discharge she was walking well and seemed healthy.

Vomiting and abdominal pain were recurrent during the next four months, and five months later they became severe and were accompanied with return of the peripheral neuritis. She was readmitted to the hospital unable to stand and with respiratory embarrassment severe enough to require the use of a Drinker respirator.

Physical examination and laboratory examination showed conditions similar to those noted previously, except that all were more pronounced. Roentgenograms of her skull showed beginning separation of the coronal sutures. Her diaphragm was completely paralyzed, and the metallic deposits in her long bones were more intense than ever. She was treated with high intake of calcium, large doses of vitamin D and intramuscular injections of magnesium sulfate. Her strength returned gradually, and she was again discharged to a convalescent home three and a half months after admission to the hospital, still unable to walk. No definite source of lead was identified. Psychologic examination at the time of her discharge showed an intelligence quotient of 87 and difficulties in learning not explained by subnormal intelligence alone.

Soon after this admission her mother died of tuberculosis in a sanatorium. The child was placed in a convalescent home, where she remained six months, and was discharged able to walk, with straps to control her toe drop. At this time roentgenograms of her long bones no longer showed lead deposits.

At 10½ years a psychologic appraisal showed an irregular mental examination, with an intelligence quotient of about 75 (Stanford-Binet). She was repeating the fourth grade and looking toward a special class or an industrial class.

CASE 13.—A boy of 18 months was admitted to the hospital because of vomiting of six weeks' duration. He was the only child of healthy parents, was born normally and did well as a baby. He held up his head at 5 months, sat up at 6 months, stood at 10 months and walked alone at 13 months. At 15 months he had a vocabulary of 3 words. He began to chew paint off his crib at 8 months, and as soon as he learned to walk he chewed it off the window sills and furniture. Vomiting had gradually increased in frequency to several times daily, and during the two weeks before admission he had exhibited strabismus and an unsteady gait.

Physical examination showed a somewhat pale baby. His fontanel was closed, and percussion of his head gave a cracked pot sound. Both optic disks were choked, and there was an alternating internal strabismus.

His blood showed a secondary anemia (hemoglobin content 55 per cent and red cells 5,400,000) and many stippled cells on smear. His spinal fluid was under greatly increased pressure and had a total protein content of 124 mg. per hundred cubic centimeters. Roentgenograms of the skull showed separated sutures, and roentgenograms of the long bones showed a dense wide band at the growing ends of the shafts of the long bones, characteristic of lead poisoning.

He was treated with intravenous injections of 20 per cent solution of dextrose and disodium phosphate and percomorph liver oil by mouth, and he was discharged in three days much improved. His family was instructed to protect him from paint and to treat him with a high milk diet and percomorph liver oil at home.

Two months later he returned to the outpatient department in good condition. His eyes were straight, but the fundi were not seen.

At 4 years he returned because of knockknees, and his mother gave a history of his having had two convulsions during the previous six weeks. At that time roentgenograms of his long bones showed some distortion of the ends of the diaphyses, but the heavy bands of lead deposit had disappeared.

He next returned, at 6 years of age, because of behavior difficulties which had been growing more pronounced since he was 2 years old. In kindergarten he had stuck a fork into another boy's face. In first grade he had been expelled for stealing pencils and pens. At home he had set the apartment on fire twice. Psychologic appraisal at 6½ years showed a mental age of 6 years and an intelligence quotient of 92. In spite of this, he could not write his name legibly or recognize any of the simplest words. On a brief visuomotor test he showed very poor visual memory and even reproduced a square as an almost circular figure.

CASE 14.—A boy of 23 months was admitted to the hospital because of irritability of one year's duration. His history, except for pica, was irrelevant; the family history was normal. He sat at 7 months, walked at 15 and talked at 18 months.

At about the time he learned to sit up he began displaying pica, eating several coats of paint off the available portions of his crib, carriage and porch railing. He also had eaten cloth, papers of all sorts, curtains, gloves, matches and pencils, and his own stools and canine stools as well. His appetite for food had suffered tremendously, and he had never eaten well. During the year before his entry into the hospital he had been extremely irritable. During the two months before entry he had become unsteady on his feet, and during the week or two before admission he had had abdominal pain and diarrhea.

Physical examination, including examination of the fundi, showed no abnormality except poor nutrition and hyperactive tendon reflexes. His blood showed no anemia, but there were numerous stippled cells on smear. Roentgenograms of the long bones showed heavy bands of density at the ends of the shafts, diagnostic of lead poisoning. His spinal fluid was not remarkable. Psychologic examination showed poor intellectual development at this time, which, taken with his early developmental history, suggested a slowing up of development.

He was treated with disodium phosphate and viosterol, and his family was instructed to guard against ingestion of lead. At home his irritability continued, as did his pica, but his parents attempted to remove all lead from his environment. He gradually improved in stability, but any infection, such as one on the dorsum of a finger, brought out the old symptoms; however, he steadily improved in language ability.

Nine months later he was again admitted because of irritability, frequent soiling and wetting of himself and a weak gait "like that of a little baby just learning to walk." His blood showed a secondary anemia but no stippling, and roentgenograms of his long bones still showed metallic deposits. An encephalogram indicated normal spinal fluid and normal ventricular outlines. In the ward his behavior improved, but his play was more like that of a 20 to 24 month old child than that of one of 32 months and his concentration and attention were poor.

The moment he returned home his behavior returned to its former low level. Vomiting and abdominal pain appeared in bouts, once severe enough to cause his admission to the hospital under suspicion of appendicitis. At 4½ years he was a problem in kindergarten, and the following year he entered the first grade, where he was having great difficulty in learning to read and write, in spite of an intelligence rating of 109.

At 6$\frac{2}{12}$ years he entered the hospital with a severe tracheobronchitis, for which intubation became necessary, and, in addition, he was treated with sulfanilamide for six days in doses maintaining concentrations of the drug between 10 and 15 mg. per hundred cubic centimeters of blood. Twelve days after the use of sulfanilamide was stopped, an agranulocytosis developed, from which he died in sixteen days.

CASE 15.—A girl of 3$\frac{4}{12}$ years was admitted to the hospital because of vomiting of three and one half months' duration. Her father had been treated for syphilis for two years. The date of his infection was unknown. Her mother was well, and there were five older siblings who were well. The oldest child had died, and the third pregnancy had resulted in miscarriage. The patient was born normally at term and did well on bottle feeding. She had had irregular amounts of orange juice and cod liver oil through infancy, but since infancy her diet had been adequate. She walked at 1 year and began to talk at the same time. At 10 months she began to exhibit pica, eating paint, bark, linoleum and dirt. Three and a half months before admission to the hospital she began to vomit from one to five or six times a day, to lose her appetite, to be constipated and to have dreams bordering on hallucinations at night. She lost 6½ pounds (2,948 Gm.) during this period.

Physical examination showed a pale, restless child with constant choreiform movements and a mild state of mental confusion. Her tendon reflexes were not obtainable.

Her blood showed a hemoglobin content of 58 per cent and a red cell count of 4,700,000. There were no stippled cells on repeated examination. Her urine was not remarkable. Her spinal fluid contained 18 white cells and 63 mg. of protein per hundred cubic centimeters. Roentgenograms of her long bones showed moderately broad metallic zones at the ends of the long bones, characteristic of lead poisoning. Wassermann and Hinton reactions of the blood and spinal fluid were negative.

She was treated with disodium phosphate and large doses of viosterol daily. Her confusion and vomiting subsided promptly, but in the ward she showed pica which was very difficult to control. She was sent to a convalescent home, where she remained two months. At first she seemed dull for her age, but in a few weeks she became responsive and attractive.

At 3$\frac{7}{12}$ years she reported to the outpatient department. At that time her right hand was so unsteady that she used the left by preference. She played with the test materials on psychologic examination like a 2½ year old child, showing special difficulties in the differentiation of shapes, though her language was adequate for a 3 year old child.

She returned to the hospital at 7$\frac{7}{12}$ years in a generalized convulsion. The mother stated that she had been well and was in the second grade in school but was unable to learn to read.

She had shown no pica since the previous admission to the hospital. The convulsion was controlled with ether and paraldehyde, and the next day, save for bites on her tongue, results of physical examination were unremarkable. Cytologic examination of her spinal fluid and blood yielded unremarkable results. Roentgenograms of her long bones showed some disturbances in growth at the ends of the long bones but no metallic bands. After recovery from the convulsion she used her left hand poorly as compared with the right for fine movement.

Psychologic appraisal revealed an intelligence quotient of 91 (Stanford-Binet). It had been 102 by the same examination a year before, with specialized defects in the field of spatial orientation. She was above the average for her age from the point of view of general information. In school work she proved totally unable to learn to read, although she could copy words readily. On the Wood Picture Completion test her associations were so simple that the results were bizarre and unscorable. She failed in all the cube designs appropriate for 6 year old children. She did picture puzzles less well than the average 6 year old child. On the Pintner-Cunningham Test no. 7 she made a great number of unpredictable errors, perseverating from one design to the next.

CASE 16.—A girl of 4 7/12 years was admitted to the hospital because of convulsions which had first appeared when she was 4 years of age. Her family, birth and past history were not remarkable. She walked at 13 months and used single words at 18 months. She had been known to chew paint, furniture and newspaper since before she was 2 years old. For some time she had had a poor appetite and severe constipation. Isolated convulsions had accompanied high fever at 4, 4 5/12 and 4 7/12 years.

Results of her physical and neurologic examinations were not remarkable. Laboratory studies showed no abnormalities save for mild secondary anemia (hemoglobin content 55 per cent). Roentgenograms of the long bones showed a broad band of increased density at the ends of the long bones, consistent with long-standing lead poisoning. A diagnosis of lead poisoning was made and treatment with vitamin D and a high calcium diet instituted.

Scattered convulsions recurred during the ensuing three years, but from 8 to 10 years of age she was free of them. At 10 years sudden recurrent attacks of gagging and hiccup developed, which were accompanied by confusion lasting a matter of minutes, and from 12 years of age she again had recurrent frank convulsions. Pneumoencephalograms showed symmetric enlargement of the lateral and third ventricles in a cranial vault of average size and were interpreted as indicative of diffuse cerebral atrophy.

Psychologic examinations were made at various ages. At 5 years her intelligence quotient (Stanford-Binet) was 90, with pronounced irregularities, and she exhibited difficulty in learning new material in spite of the adequate rating. At 8 years similar results were obtained. At 12 7/12 years she gave a very poor and irregular performance on the Terman test, her results indicating a mental age of 8 years and an intelligence quotient of 65. She could not deal with the picture absurdities or comprehension at the 7 year level, while she succeeded with two items of comprehension at the 9 year level. On the Wood Picture Completion test she did as well as the average 9 year old.

In dramatic contrast to her low rating she attained fifth grade score in most subjects on the Metropolitan achievement tests. Although her actual test performance was so confused that she characteristically failed more tests than she passed, many of her failures were on the easier tests, while some of her successes were in fractions and decimals.

The school reported that in the sixth grade she was a desirable pupil and worked conscientiously under a slight strain to keep up her grades. Her mental capacity was so unpredictable that it was felt that plans for her education should be formulated more with reference to her teachers' attitudes than to the child's actual ability to keep up.

During the past winter she has been out of school for ten weeks because of appendicitis.

CASE 17.—A girl of 1 11/12 years was admitted to the hospital because of convulsions and a rash. Her mother had observed that she was chewing paint at 1 year and 10 months. One week later, in connection with fever, she had a convulsion which left her exhausted for several days. Her local physician demonstrated stippled cells in her blood at this time and referred her to the hospital, where roentgenograms of the long bones showed dense bands at the end of the diaphyses, consistent with lead poisoning.

After that she remained well as far as her family knew, except that she vomited easily and often. When the vomiting was severe, at 5 years, her local physician again found stippled cells in her blood and treated her successfully with calcium and vitamin D.

At 5 years and 3 months she displayed frequent lapses of consciousness lasting one or two minutes, sometimes accompanied by falling and involuntary voiding and usually followed by sleepiness for some minutes.

Pneumoencephalograms at this time showed normal ventricular outlines. Roentgenograms of the long bones showed no evidence of lead, though stippled cells had been found three months before.

Psychologic examination at 6 years, when she was in the first grade, showed irregularities of intellectual development. On the Stanford-Binet test her intelligence quotient was 94. She had a poor memory for stories, and after nearly a year in school she had not learned to write her name or make figures, though she had learned to read.

CASE 18.—A boy of 10 months was admitted to the hospital because of irritability and vomiting of three days' duration. He was born by forceps delivery, and, because he spit up blood on the first postnatal day, he received one transfusion. He was breast fed up to the time of admission to the hospital, and his mother had used lead nipple shields throughout his life. At 10 months he sat up with assistance. Three days before admission he became irritable, vomited projectilely and complained of abdominal pain. These symptoms continued to the time of admission.

On physical examination he was pale, his fontanel was bulging and there was a slight degree of edema of both optic papillae. His temperature ranged between 102 and 103 F. for two weeks.

Laboratory examination revealed a mild secondary anemia (hemoglobin content 70 per cent and 3,300,000 erythrocytes) and 2 to 5 stippled cells per oil immersion field on smear.

His spinal fluid was xanthochromic, was under increased pressure and showed a total protein content of 160 mg. per hundred cubic centimeters and 12 mononuclear cells per cubic millimeter.

Roentgenograms of the long bones showed dense bands at the end of the diaphyses, and roentgenograms of the skull showed separation of the cranial sutures.

He was treated by intravenous injections of 25 per cent solution of dextrose, intramuscular injections of magnesium sulfate and large doses of viosterol in oil.

His irritability diminished slowly, and his blood pressure remained about 120 systolic and 100 diastolic. During the third week a transient right wrist drop was noted. Even at the end of his three and a half week stay there were still a few stippled cells to be found in blood smears. He was discharged with the statement that a good deal of cerebral damage had probably been done.

A month later his blood pressure was 90 systolic and 40 diastolic, and he seemed happy and cheerful. At 14 months he was standing and saying "dada," and at 16 months he was noted by one of us (E. E. L.) to be acting like a 10 to 12 month old baby. At 19 months he seemed to all observers except the psychologist to have caught up with other babies of his age, but on psychologic examination he showed difficulties in dealing with space and direction, which persisted to the age of 5 years. His intelligence quotient at that time was 90.

The boy entered first grade at the age of 6 years and was promoted each year. At 7 years he had an intelligence quotient of 103. There was no evidence of visuomotor disability, and he could read and write well for his age.

CASE 19.—A 2 year old boy was admitted to the hospital because of weakness and limping of his left leg of one week's duration. He was delivered by forceps after a three day labor. He was weak after delivery and would not nurse. At 2 weeks of age he was taken to the Massachusetts Eye and Ear Infirmary, where he was treated for four weeks because of discharging eyes. He was discharged considerably below his birth weight, but at home he did well on formulas prescribed by another hospital (the nature of the formulas was not stated). Cod liver oil and orange juice had been taken in adequate amounts from early infancy.

He lived with his family in an old nine room house, in which the piping was entirely of lead. His father was a painter. The patient had been known to play with paint left about the house, though he had not been known to eat any.

His development was average. He held up his head at 3 months, sat up at 6 months and walked at 14 months. He was not talking, however, at 2 years.

A week before his admission to the hospital the patient showed a disinclination to walk and began to drag his left leg. The weakness became gradually more pronounced.

Physical examination revealed nothing remarkable save that in walking the left foot was not raised off the floor; the leg was rotated externally, and the foot dragged forward. The tendon reflexes were lively and approximately equal on the two sides; perhaps the reflex on the left side was somewhat livelier. The plantar reflexes were normal.

His blood showed a secondary anemia, with a hemoglobin content of 50 per cent by the Tallqvist method and a red cell count of 4,800,000. Several smears failed to show stippled cells.

Roentgenograms of the long bones showed dense bands at the ends of the shafts consistent with lead poisoning.

He was discharged unchanged after three weeks, on a high calcium diet with cod liver oil (3 drachms [11.7 Gm.] daily). No investigation of the water was made, but the parents were warned of the danger of allowing him to eat paint. A month later he returned to the outpatient department and was regarded as well.

A year later he returned because of pallor, irritability and loss of appetite. At this time he was definitely known to chew paint off objects, though his family tried to prevent him. The weakness of the leg had not recurred.

Physical examination disclosed nothing remarkable except pallor, which was confirmed by a hemoglobin content of 50 per cent, a red cell count of 2,600,000 and a smear showing moderate achromia but no stippled cells. Roentgenograms of the long bones again showed dense bands at the growing ends of the shafts. He was discharged on a high calcium diet with iron and ammonium citrate and cod liver oil (3 drachms [11.7 Gm.] daily). In a month his hemoglobin content was 80 per cent and his red cell count 4,000,000.

At 12 years he returned to the outpatient department because of enuresis. He gave a general impression of being dull. His mother gave the following report of his school progress: "He started to school at the age of 5 years. He spent two years in the first grade. The next year he had a teacher at home. The following year he spent in the second grade. He will start the fifth grade in the fall." Psychologic tests have not been given.

CASE 20.—A girl of 3 years was admitted to the hospital because of vomiting and abdominal pain of three months' duration. Her father and mother were separated at the time. Her birth and early development had been normal. She walked at 13 months and talked at 14 or 15 months. From the time she began to move about she chewed anything and everything, including paint from her crib and from window sills. About three months before her admission to the hospital she began to vomit, and the vomiting increased in frequency. A month later abdominal pain, irritability and pallor supervened. One month before admission strabismus was noted. During this period a gradual loss of weight occurred. She had complained of headache during the few days before admission.

On physical examination she was dull and drowsy. Her optic disks were noticeably choked, and her skull was hyperresonant on percussion. Her tendon reflexes were everywhere hyperactive, and Babinski signs were elicited on both sides.

Her hemoglobin level was 50 per cent; her red blood cell count was 2,600,000, and her blood smear showed achromia and 8 to 10 stippled cells per high power field. Her spinal fluid was under increased pressure and contained 18 cells per cubic millimeter and 108 mg. of total protein per hundred cubic centimeters.

Roentgen examination showed heavy bands at the growing ends of the long bones, consistent with lead poisoning, over a long period of time, with an especially heavy recent deposit.

Increasing drowsiness and vomiting continued in spite of treatment with hypertonic solution of dextrose (20 per cent) and dibasic sodium phosphate, and on her fifth hospital day bitemporal decompression was performed.

Frequent lumbar punctures were performed, with the removal of large amounts of spinal fluid (up to 200 cc. on one day), during the next four weeks. After that the patient gradually became interested in her surroundings, her vomiting stopped and she made a rapid recovery during the last two weeks of her stay. Six weeks after admission she was playing "normally" in bed, but psychometric examination showed a general level of intelligence around 3 rather than approaching 4 years, with special disabilities in the field of spatial relationship. She was discharged on the fifty-first day in the hospital, weak and underweight but able to walk and showing an active interest in life.

In spite of her mother's efforts she continued to eat paint at home, stripping all the paint off the porch railing before she was caught. A year later she was again admitted because of vomiting and abdominal pain. Her decompression wounds were bulging, but her optic disks were normal. Her gums showed a lead line. Her hemoglobin content was 53 per cent and her red blood cell count 3,900,000, with marked achromia and many stippled cells on smear. Spectroscopic examination of the blood showed a level "very much above anything found in normal controls." Roentgenograms of her long bones showed wide bands of lead deposit with recent accentuation at the growing ends. Roentgenograms of her skull showed no evidence of pressure. A lumbar puncture showed fluid under apparently increased pressure, with 6 monocytes per cubic millimeter and a total protein content of 46 mg. per hundred cubic centimeters. She was discharged on a high calcium diet and 18 drops of combined fish liver oils daily, and with assurance that she would be protected from paint in the future.

She was next seen when she was 9 years and 6 months of age, in response to a follow-up letter. Although she was friendly and outgoing, she was doing badly in school in the third grade, had a mental age of 7 11/12 years and an intelligence quotient of 80, with special defects in spatial orientation. Her score on the Ellis visual designs test was 1.5, which was below the calibrated level for her age.

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