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ADDENDUM

Subsequent to the submission of this paper for publication, Dr. J. Godwin Greenfield of London, England, brought to the attention of one of us (W. L. D.) the report by Collins and Armour* in 1912, of an additional case of fatal post-mumps encephalitis. This concerned a boy 11 years of age who developed the first signs of encephalitis seven days after the onset of mumps. After three days of rather minor complaints of dizziness, he rapidly deteriorated and died twelve days after the onset of mumps. Accompanying the report are beautiful photomicrographs of typical perivascular lesions.

*Collins, J., and Armour, D. G.: Acute Bulbar Palsy Following in the Wake of Mumps, Rev. Neurol. & Psychiat. 10: [illegible], 1912.

THE LATE EFFECTS OF LEAD POISONING

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THE late effects of lead poisoning on the physical and psychological development of children have received little attention. Though thousands of articles have appeared on lead only, Byers and Lord,[1] Ebbecke,[2] and Goettsch and Mason[3] have reported on the final effects of lead poisoning in patients followed for a variable period of time. The sequelae of lead intoxication in children appear to be mainly psychological. Little interference in physical development has been reported. However, two of our patients showed optic atrophy; one with permanent blindness.

During the ten-year period, 1940 through 1949, twenty-six children were treated for lead intoxication. The source of the lead was either from paint, plaster, or lead toys. Seven children (27 per cent) died, six on initial hospitalization and one two years later at another hospital with "lead encephalitis." Eleven of the remaining nineteen patients have been followed for a 5- to 10-year period during which repeated physical and psychological evaluations have been carried out, including roentgenological and chemical lead determinations. Five of the eleven patients were diagnosed and treated on an outpatient basis and the other six were hospitalized. Two of the former group were subsequently hospitalized with intercurrent infections which precipitated a return of the symptoms of lead intoxication.

To estimate the ultimate effects of lead poisoning on body and mind, an accurate evaluation of the original degree of lead encephalopathy is desirable. From the data in Table I, the hospitalized patients were empirically classified as mild, moderate, moderately severe, and severe. There is no concrete basis for such an evaluation. Blood and urine lead determinations vary with initial therapy (Cases 4 and 5). The severity of the central nervous system manifestations appears to depend on duration of lead intoxication, manner of exposure, and precipitating illness (Case 1). Details of such factors are often difficult to elicit accurately from the parents.

The symptoms in the five outpatients were vague, nonspecific, mainly gastrointestinal, and of variable duration. They are listed in order of frequency as follows: vomiting (four cases), constipation (three cases), malaise (three cases), anorexia (two cases), pallor (two cases), diarrhea (one case), irritability (one case).

The signs, symptoms, physical and laboratory findings in the six children who were hospitalized for lead encephalopathy are listed in Table I.

Except for the convulsions, all the other symptoms exhibited by the

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TABLE I

CASE	AGE (YR.) RACE SEX	CONVUL. SIONS	GENERAL	NEUROLOGICAL	LABORATORY	X RAY COMPATIBLE WITH METAL POISONING	SEVERITY
1	2 ♀ W	1	Malaise Anorexia Fever Pharyngitis	Irritability	Normal blood and urine	+	Mild
2	2½ ♀ W	1	Vomiting Constipation	Hemiparesis Papilledema	Basophilic stippling. Urine lead 0.93 mg. L. CSF pressure elevated. Protein 62 mg. 100 ml.	+	Moderately severe
3	2½ ♂ W	0	Vomiting	Weakness Lethargy "Meanness" Papilledema	Basophilic stippling. EEG indeterminate for age. CSF 575 mm. pressure. Protein 254 mg. 100 ml	+	Severe

Neurosurgical decompression 18 hours after admission with continuous spinal drainage for 18 to 72 hours to prevent herniation through subtemporal decompressions.

4	2 ♀ W	0	Anorexia Pallor Constipation	Marked irritability and weakness	Basophilic stippling. EEG not disordered Urine lead 0.227 to 1.2 mg. L. CSF 300 mm. pressure. Protein 198 mg. 100 ml	+	Moderate
5‡	3 ♂ W	1	Anorexia Vomiting	"Behavioral difficulties"	Blood lead 1.134 mg. 100 ml. EEG disorganized with paroxysmal bursts. CSF normal	+	Mild
6	2 3/12 ♀ W	0	Anorexia Malaise Pallor	Papilledema, associated with "spitting" cranial sutures	Basophilic stippling. EEG moderately slow dysrhythmia CSF Cardiac pressure Protein 72 mg. 100 ml		Moderate

*Normal values below 0.05 mg. 100 ml
†Normal values below 0.09 mg.
‡One of identical twins, both having lead poisoning.
Patient 5 also had hay fever—asthma, not developed to marked proportions.

TABLE II

SYMPTOMS	NUMBER OF PATIENTS	PERCENT	DURATION	AVERAGE
Irritability	8	72	2 wk.–6 mo.	1.2 mo.
Vomiting	4	36	6 wk.–6 mo.	1½ mo.
Constipation	4	36	1–2 mo.	1 mo.
Anorexia	3	27	1–2 mo.	1 mo.
Malaise	1		1 1/2 yr.	
Pallor	2	18		2 wk.
Abdominal pain	2	18		2 wk.
Convulsions	3	50	Intermittent	
Papilledema	3	50	Rapid decrease 24–48 hr.	
Basophilic stippling	1	66	Disappeared 48–72 hr.	

eleven patients persisted for variable periods of time after therapy as noted in Table II.

On recent physical examination all of the children were of normal height and weight for age except one with exogenous obesity. Two of the children (18 per cent) had bilateral optic atrophy and a third had a high degree of myopia.

The x-ray findings of the long bones of the eleven patients are presented in Table III. Like the symptoms in lead poisoning, the roentgenographic changes showed a variable degree of persistence. In the entire series of twenty-six patients, complete disappearance of the "lead line" at the distal end of the shaft was eventually noted with recurrence six to seven months later in two patients. These two patients were proved to have reinstituted the paint-chewing habit. Three patients had x-ray descriptions of transverse "linear distal densities" 2 or 3 mm. from the ends of the shaft interpreted as "minimal evidence of bone growth retardation."

Urine and blood lead determinations contributed nothing to the follow up study.

Electroencephalograms were obtained in six of the eleven patients. There was no consistency in regard to the time of the initial or follow-up electroencephalogram as related to the time of initial lead intoxication. Cases 1, 2, 9, 10, and 11 had none done. Case 3 initially was indeterminate for age, but two years later the record was consistent with organic brain disturbance. Case 4, in 1948, showed a record consistent with the age of the child. In 1954, the child had several grand mal seizures, but then became well controlled on small doses of phenobarbital. In Case 5, repeat records showed paroxysmal slow dysrhythmia for two to three years, a posterior triangle focus also being noted on the latest examination. The records of this patient have shown the greatest disorganization of the entire series and the child needs psychiatric guidance at the present time. The remaining Cases 6, 7, and 8 were almost identical in showing definite slow dysrhythmia of a variable degree.

The treatment of all patients was identical. Sodium citrate alone or with citric acid was given to maintain a slightly elevated CO_2 content (28 to 30 meq. per liter). The dosage varied from 3 to 6 Gm. daily in divided doses, for a period of from

TABLE III. RADIOGRAPHIC FINDINGS IN LONG BONES

CASE	ONSET OF ILLNESS	FOLLOW UP: 1950	1954
1	1941	0	0
2	1948	0	0
3	1948	1	0
4	1949	0	0
5	1949	0	0
6	1949	0	0
7	1949	0	0
8	1949	0	0
9	1946	1	0
10	1950	0	0
11	1949		0

two weeks to two and one-half years.[5, 6, 8] A summation of the follow-up is presented in Table IV.

PSYCHOLOGICAL EVALUATION

Byers and Lord note in their study of twenty children who had lead poisoning in early infancy that despite normal motor development during infancy only one child lived up to the promise of his early development. In a few children successive psychological examinations showed significant drops in intelligence quotients. These drops were thought to be the result of failure of mental development rather than actual mental deterioration. As the chronological age advanced, mental growth did not keep pace with it. In others, however, the intelligence quotient remained well within normal limits. A specific failure of development in the sensorimotor sphere was the outstanding finding. Byers and Lord felt that the lead in the circulation of an infant 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.

Along with the dilatory effects of mental growth, a high percentage of behavior difficulties were recorded by Byers and Lord. Such difficulties arose from an inward driveness which they classified as "forced reaction to stimuli in the environment." Strauss and Lehtinen,[7] Bender,[7] Bakwin,[9] and Stryker[10] consider this as evidence of cortical damage independent of etiology. Klebanoff and associates[11] describe the brain injured child as hyperkinetic, overactive, restless, impulsive, and uncoordinated, but by

TABLE IV. SUMMATION OF FOLLOW-UP

CASE	LENGTH OF TIME SINCE EXPOSURE (YRS.)	INITIAL SYMPTOMS	PHYSICAL SEQUELAE
1	10	Encephalopathy, convulsions, coma	Obesity, enuresis
2	6	Convulsions, drowsiness, encephalopathy, right hemiparesis	
3	6	Lethargy, vomiting	Bilateral optic atrophy
4	5	Irritability	Convulsions
5	5	Encephalopathy, convulsions	Abnormal EEG, behavior problem
6	5	Mild gastrointestinal symptoms. Treated as outpatient 5-49. Hospitalized 11-49 with gastrointestinal symptoms	
7	5	Encephalopathy, weakness	Bilateral optic atrophy
8	5	Gastrointestinal symptoms. Treated as outpatient	Decreased vision
9	5	Gastrointestinal symptoms. Treated as outpatient	
10	5	Gastrointestinal symptoms. Treated as outpatient. Hospitalized 6 mo. later with pneumonia 7-49	
11	5	Gastrointestinal symptoms. Treated as outpatient	High degree of myopia

pothesize that cortical maturation with compensatory development of function can occur since a less differentiated infantile cortex is involved, negating the influence of an injury which in an adult would have serious long-term sequelae.[12]

The psychological aspect of the present study was undertaken to evaluate systematically the residual psychological effects in a group of children who had had proved lead intoxication in late infancy. No records of original intellectual functioning were available prior to the poisoning. Normal mental and motor developmental progress previous to the lead intoxication was assumed in each case because of the past histories given by the parents.

Psychological testing was done in 1950 with re-evaluation in 1954. The time of the initial testing ranged from immediately after the child recovered from the lead intoxication to five years after the toxic state. Because of the immaturity of the group at the first testing it was possible only to obtain measures of intellectual and social development (Stanford-Binet intelligence scale[13] and the Vineland social maturity scale[14]).

PSYCHOLOGICAL RESULTS

Reference to Table V shows that at the time of the initial testing only two children had overall intelligence quotients below the dull normal level, and two were in the bright average to superior categories. One of the patients below the dull normal level, Case 4, was a hyperactive, irritable child who was extremely difficult to test, and the examiner felt that the I.Q. measure of 59 was minimal. At the time of the second testing none of the children were below the dull normal level of intelligence and four were

TABLE V. COMPARISON OF RESULTS OF INTELLIGENCE TESTING IN 1950 AND 1954, OF SOCIAL MATURITY IN 1950 AND 1954, AND GOODENOUGH I.Q. IN 1954 FOR ELEVEN CHILDREN WHO HAD LEAD POISONING

CASE	AGE AT ONSET (YR.)	1950 CHRONOLOGICAL AGE (YR.)	BINET I.Q.	INTERMEDIATE TESTING	1954 CHRONOLOGICAL AGE (YR.)	BINET I.Q.	GOODENOUGH I.Q.	1950 VINELAND I.Q.	1954 VINELAND I.Q.
1	2	7.4	106		11.5	109	65	115	111
2	$2\frac{1}{2}$	7.1	86		8.1	88	86	116	102
3	$2\frac{1}{2}$	1.3	Appeared normal		8.3	99	Blind	87	83
4	2	3.1	59	74	7.1	88	74	76	100
5	3	3.3	127	137	7.6	110	73	73	105
6	$2\frac{1}{2}$	2.1	100		6.6	122	112		127
7	$2\frac{1}{2}$	3.1	82		7.3	87	83	95	105
8	$2\frac{1}{2}$	3.1	78		7.1	84	98	89	98
9	2	3.1	97		7.3	99	99	105	105
10	2				6.3	111	91		111
11	$1\frac{1}{2}$	2.6	116		6.7	125	90	116	105
Mean	2.05	3.1	93.77		7.6	102.00	85.1	99.11	105.00
Standard deviation			18.37			13.76	1.08	13.71	10.31

Rank order correlation between 1950 and 1954 I.Q.'s .08

T Test between 1950 and 1954 I.Q.'s (corrected for correlations) 2.12*

Rank order correlation for Binet and Goodenough I.Q.'s 1954 .05

T Test for Binet and Goodenough I.Q.'s 1954 (corrected for correlations) 3.28†

*Significant at .05 level of confidence.
†Significant at .01 level of confidence.

in the bright average to superior categories. The bright average to superior children showed little unevenness in their functioning. The type of items most frequently failed by the children in the dull normal category were items involving abstract thinking. Although this is a characteristic failing of children with brain damage, it is also the type of thinking which would be expected of children with dull normal intelligence natively. Three of the eleven children showed some difficulty copying a diamond at the seven-year level, but there was no conclusive evidence in the over-all intellectual functioning characteristic of children with brain damage.

It is recognized that intelligence tests, made at different times on the same individual, may vary as much as ten points on the basis of chance factors alone. Reference to Table V shows that I.Q. changes in three of the eleven children (Cases 4, 5, 6) were significantly greater than would be expected by chance. Two of these showed improvement, while one went down. However, with the exception of Case 5 who decreased, all of the other children showed an increase of from 2 to 9 I.Q. points. The difference between the average I.Q. of 94 at the time of initial testing and 102 at the time of the last testing shows an improvement in intellectual level which is significant at the .05 level of confidence (would be expected to occur on the basis of chance only five times out of 100). A continuing intellectual decline after the initial testing, as expected from the results of Byers and Lord, was not found in our series. Instead, there was some evidence of an actual increment in intellectual functioning.

Even greater significance is added to this finding when it was realized that the one child who showed a decrease in intellectual functioning was under psychiatric care. A complete psychological study on this boy three months previously was summarized as showing: "Severely neurotic personality structure characterized by marked anxiety, regressive and repressive trends in a child of superior intelligence (I.Q. 126). Emotional problems are affecting functioning and relationships. There are also some organic indications which need further evaluation. Treatment assets are: superior intelligence, ability of ego to maintain itself despite stress, conformity and the anxiety itself."

It seems possible that the decline in functioning ability in this one case was due, at least in part, to a functional psychological disturbance.

On both testings, social development, as measured by the Vineland Social Maturity, was somewhat above intellectual development. Although these differences were not significant, the suggestion is present that these children are somewhat better able to perform in practical everyday situations.

On the second testing, however, the intellectual measure obtained from the Goodenough draw a man test was significantly lower than that obtained from the Stanford Binet intelligence scale at the same time. The difference between the average Goodenough I.Q. of 85 and the Binet I.Q. of 102 was significant at the .01 level of confidence. Though the drawing test primarily measures visual motor functioning, while the intelligence scale is primarily a verbal test,

the significant positive correlation which has been demonstrated between these two tests indicates that they measure two types of behavior which tend to develop at similar rates. The correlation of .05 between these two other words, it would seem that while the over-all intelligence of these children remains intact and is developing normally, a definite deficit is seen in their visual-motor functioning. Furthermore, qualitative analysis of the

TABLE VI. 1954 GRADE PLACEMENT, SCHOLASTIC ACHIEVEMENT, AND RESULTS OF VISUAL-MOTOR TESTS OF BRAIN DAMAGE OF ELEVEN CHILDREN WHO HAD LEAD INTOXICATION IN INFANCY

CASE	GRADE	1954 EDUCATIONAL LEVEL	1954[a] WIDE RANGE ACHIEVEMENT READING	ARITHMETIC	GRAHAM-KENDALL CATEGORY	BENDER GESTALT
1	5	Failed second grade. Doing well now except arithmetic	6.0 grade	3.6 grade	Borderline	Rotated designs, A and 3; difficulty with spatial relations
2	2	Held back ½ grade	2.5	2.3	Normal	Normal
3		Blind, in school for blind	Blind	Blind	Blind	Blind
4	3	School problem	1.1	1.1	Brain damaged	Reversed A; rotated 8; primitive loops and lines; difficulty with angulation
5	4	School problem	2.1	2.3	Brain damaged	Rotated designs A, 3, 4, 5, 8; primitive loops, difficulty with angulation
6	2	Does well	1.2	1.1	Borderline	Rotated designs A, 3; perseverated on 1 and 2; primitive loops, difficulty with angulation
7	1	Held back 1 grade	1.0	1.1	Brain damaged	Perseveration; poor spatial relations, difficulty with angulation on 7 and 8
8	1	Held back ½ grade	1.3	1.3	Brain damaged	Primitive loops and lines; poor spatial relations; difficulty with angulation on 7 and 8
9	2	Held back ½ grade	1.6	1.5	Brain damaged	Rotated designs A, 4, 5, 7; difficulty with angulation on 7 and 8
10	1	Does well	0.9	1.1	Normal	Perseveration in design 2, primitive loops and difficulty with angulation
11	2	Does well Poor in drawing	2.3	2.7	Brain damaged	Spatial relations poor on a 2, 4; difficulty with angulation on 7 and 8

[a] Reported to be doing well in school.

measures for this group would indicate that there is no relationship other than chance between the developmental rates of these two types of behavior for these children. In drawings revealed the disorganization in body image and immature drawings which Silver[16] describes as characteristic of children with brain damage.

It is of interest, then, to note, that despite intellectual functioning within the normal range, only three of the eleven children are reported by their mothers to be doing well in school. (See Table VI, Cases 6, 10, and 11.) Two of these patients (Cases 6 and 10) show academic achievement below their grade placement, which suggests that they are actually having more difficulty in school than has been reported by their parents.

Tests of visual-motor functioning which are specifically designed to measure behavior characteristic in the individuals with cortical damage reflected considerable deficit in this area. On the Graham and Kendall[17] visual motor test with correction for chronological age, Cases 2 and 10 were within normal category. Cases 1 and 6 were in the borderline brain-damaged category, and Cases 4, 5, 7, 8, 10, and 11 showed visual-motor deficit within the brain-damaged category. At the extreme end of the continuum of visual motor deficit was Case 3 who is totally blind as a result of the lead poisoning.

An even more pronounced deficit was noted in the group's performance on the Bender-Gestalt visual motor test.[18] Case 2 was the only child who was able to copy the designs of this test in a fashion which was normal for her age level. All of the other children showed the rotation of designs, perseverations, difficulty with angulation, and substitution of primitive loops and lines for dots which Silver[18] describes as characteristic of children with brain damage.

Case 2 is the one child in this group of eleven, then, who does not show a visual-motor deficit. This is of particular interest when it is remembered that her toxic reaction was one of the most severe, and at the time of her hospital admission she suffered a left hemiplegia. This suggests that there is no direct correlation between the severity of the toxic reaction and the severity of the residual effects reflected in the psychological functioning.

A closer consideration of the type of errors made by these children in their visual-motor functioning shows behavior which is paralleled in their academic achievements. The rotations of their drawings of designs placed directly in front of them suggest that these children would find it difficult to learn to read in a teaching situation which requires them to recognize the visual picture rather than paying attention to the phonetic elements of the word. This was borne out in their reading tests by a considerable number of reversed letters, reversal of whole words, and of misrecognition of words due to attention to the first letters.

DISCUSSION

Thorough physical and laboratory examination revealed little that could be attributed to the late effect of lead intoxication in a group of children followed for a five- to ten-year period of time. The effect of increased intracranial pressure in Case 3 resulted in blindness as the only physical residuum in the whole group.

However, the organic brain damage that resulted is only apparent in the specialized psychological tests done repeatedly, and the results are quite similar to those seen in children with brain damage due to birth injuries or more specifically, cerebral anoxia

From previous pathological studies of our own and others, serious vascular damage, cerebral edema, with resulting cortical injury, is the underlying basis for the picture of lead encephalopathy.[18] The intellectual degradation as reported by Byers and Lord is not confirmed and the outlook should be definitely more hopeful.

It would seem that these children with visual-motor deficit are placed in an extremely frustrating position during their primary grades. Though their learning capacities are within normal range, the visual-motor deficit presents a serious handicap to the child in the present school system. The presentation by visual stimuli for learning is frustrating. Special tutoring in this one area or placement in a school situation which would allow the use of other sense modalities in learning, such as phonetic or kinesthetic approaches, might minimize this frustration.

Case 1 is presented in support of this hypothesis. Though the pathology in his home situation cannot be denied, the intermediate testing prior to entrance to school yielded an IQ of 137. During three school years, his teachers complained that he was "a negativistic, restless, poorly attentive child, who refused to learn." Because of such behavior and because of the general family situation, psychiatric help was advocated. The extremely frustrating situation presented by a boy of superior intelligence who is handicapped in learning by poor visual motor coordination cannot be overlooked. This handicap might be a contributory factor in his need for psychiatric help.

The pictures presented by Cases 1 and 3 are more hopeful and suggest that, with understanding help, the children may learn to adapt to the deficit. Case 1 is the oldest child in the series and her lead intoxication occurred ten years prior to the last evaluation. She has attended parochial school and has received a good deal of individual attention. The primary grades presented difficulties characteristic of any brain-damaged child and there was failure to pass the second grade. Her mother reports that she is doing well in school now, but is slow in arithmetic. Her achievement test scores corroborate this, as she is at her grade level in reading but is retarded in arithmetic. The visual-motor deficit is not as pronounced as in many of the other cases. This suggests that the effects of cortical injury may be negated as the central nervous system matures. Patient 3 is totally blind and has been receiving training at the Missouri State School of the Blind. Neither difficulties in the primary grades nor the behavioral troubles are as marked as in the other children.

At the time of the initial testing five of the children presented the driven, negativistic, hyperactive behavior which is characteristic of children with brain damage. None of the children displayed this type of behavior at the time of the final examination. Cooperation was excellent, even with the boy who is under psychiatric care. It was only when his mother entered the room that his negativism was evidenced. The one common personality trait exhibited by the group was that of strong motivation to achieve in all tasks, with repeated voicing of fears that they were

not doing well or might fail. Such dissipation of hyperactivity or inward driveness with cortical maturation associated with some types of brain damage concurs with previous reports by other authors.

The attitudes of the mothers toward these children were of interest. The fact that eleven mothers out of a possible nineteen brought their children in for re-evaluation is an evidence of their conscientiousness. In most of the cases there appeared to be an overprotective attitude. The mother of Case 1 wondered if she "babied her daughter too much" and feared the family was overprotective. The child is obese and enuretic. Case 2's mother has showered an unusual amount of attention on her child "because she has been so sick." The child's teachers complain that "she acts up in school and refuses to obey," although the mother denies this problem at home. The mother of Case 5 wondered if her son's fearfulness, excessive sense of guilt, and "sissy behavior" were the result of lead intoxication and admitted the need of protecting him because of his lack of assertiveness.

The parents naturally assume a certain amount of responsibility for the lead intake and resulting lead intoxication. Such responsibility is associated with consequent guilt feelings and is reflected in the overprotectiveness of many of the parents in this series. Pediatric counseling of the parents of children with lead intoxication should help them with their subsequent attitudes and do much to prevent the development of additional personality problems. Realization of the visual-motor deficits in these children should be of aid to their teach[ers] and prevent to a certain extent [the] development of problems as [are] described.

SUMMARY

Eleven cases of lead intoxi[cation] have been followed for a peri[od] from five to ten years with [re]physical, psychological, and l[ab]tory examinations.

The physical sequelae consist[ed] blindness and cerebral dysrhy[thmia]

Sodium citrate was the s[ole] therapy in all cases and was [given] over a variable period of time [and] there was no laboratory evide[nce] lead intoxication.

There is no evidence in this [series] of continuing mental deteri[oration] Considering the major mode[rn] struction in the primary grad[es] felt that these children are at [a] ous disadvantage and should [be] special attention to offset their [gross] motor deficit. As the child [grows] there is a gradual loss of the [over]driven hyperactive behavio[r] quently seen in brain-damage[d] dren. There is no direct co[rre]between the severity of the illn[ess] the amount of residual effect.

Pediatric counseling might [be] fluential in reducing a pare[ntal] overprotective attitude which [is] the child's problems.

The authors would like to gra[te]knowledge the assistance of Wm. [Al]ner, M.D., Edward Pearcy, M.D., [and] Caldwell, Ph.D., in the preparation [of] report.

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A Problem in Neurosurgery

...er 1641. One of the deacons of Boston church, Jacob Eliot had a daughter ... of age, who being playing with other children about a cart, the hinder ... fell upon the child's head, and an iron sticking out of it struck into the ... and drove a piece of the skull before it into the brain, so as the brains came ... Seven surgeons (some of the country, very experienced men, and others of the ... rode in the harbor) being called together for advice, etc, did all conclude, the brains, (being about half a spoonful at one time, and more at other that there was no hope of the child's life, except the piece of skull could be But one of the ruling elders of the church, an experienced and very skilful ... not to take that course, but applied only plasters to it, an withal earnest ... made by the Church to the Lord for it, and in six weeks it pleased God that ... skull consumed, and so came forth, and the child recovered perfectly; nor the senses at any time.

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