hindbrain or else the entire brain stem in scattered fashion bilaterally. In these cases the signs were generally slight or moderate compared with the rest of the picture, and they related chiefly to the midbrain without evidence of involvement of the cranial nerve nuclei below the abducens.

Alcoholic polioencephalitis superior may be distinguished from these cases by its character as a deficiency disease: the alcoholism is outstanding, and there are signs of general malnutrition. Changes in the skin and mucous membranes bear witness to lack of specific vitamins. The mental picture is characterized by restlessness or somnolence and often with disorientation. Lastly, apart from the oculomotor disorders and ataxia, localizing neurologic signs are not prominent.

To summarize, the condition in the cases reported was characterized by a history of trauma of the head, a background of alcoholism (predisposing to trauma of the head), the development of signs over a period of days to a few months and a variable and fluctuating course. All together, these features go to make up a clinical picture consistent with subdural hematoma and certainly warranting the suspicion of that condition. And, when that diagnosis is even suspected, the presence of signs of disease of the brain stem should not be considered to contraindicate exploratory trephination.

Notes on Paralysis of Gaze.—A word of caution is in order with regard to the diagnostic significance of oculomotor signs. It is not sufficiently recognized that these signs are not entirely specific and that impairment is sometimes manifest as the result of generalized depression of the sensorium. Kestenbaum [28] has pointed out that any gaze movement involves various levels of activity. Hence in stupor there often occur nonspecific disturbances of these functions. Upward gaze is the first to suffer, followed by convergence, and in deep coma no gaze movements whatever may be elicited. In the review, already referred to, of 48 cases in the Bellevue neurologic service between 1932 and 1940, there was a fairly high incidence of all kinds of oculomotor disturbances in stuporous and semicomatose patients, and a similar incidence obtains with respect to a number of published series. I have tried to select only those cases in which the level of cooperation was such as to warrant the analysis of eye movements.

CONCLUSIONS

In cases of chronic subdural hematoma there occasionally appear signs referable to disease of the brain stem. Because radically different approaches to therapy depend on the appropriate diagnosis in such cases, and particularly because timely surgical intervention is of such dramatic benefit in subdural hematoma, it is important that this fact be kept in mind and that indications for exploratory operation be considered accordingly.

When the other clinical findings warrant the suspicion of subdural hematoma, the presence of signs of involvement of the brain stem should not stand in the way of operative exploration.

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28. Kestenbaum, A.: Blickbewegungen und Blicklähmungen, Confinia Neurologica **2**: 9, 1939.

INCIDENCE OF LEAD POISONING IN THE CITY OF BALTIMORE

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Since 1935 the Bureau of Laboratories of the Baltimore City Health Department has provided a routine laboratory service for the quantitative estimation of lead in blood and other body fluids as an index of abnormal lead absorption in cases of suspected lead poisoning.[1] The cooperation of those physicians and hospitals who made use of this service was solicited in order to make available the clinical histories of the persons whose bloods were examined for lead. In this way considerable information was obtained about the local occurrence of both industrial and nonindustrial lead poisoning.

A summary of the incidence of lead poisoning in the city of Baltimore during the period 1931-1940 is shown in table 1. These data were obtained from the following sources: (1) reports of occupational lead poisoning, (2) reports of deaths from lead poisoning and (3) cases ascertained in a follow-up of the blood lead laboratory service.

TABLE 1.—*Lead Poisoning in Baltimore, 1931-1940*

Year	Total Cases	Fatal Cases		Nonfatal Cases	
		Adult	Child	Adult	Child
Total	229	7	40	66	89
1931	3	1	2	...	...
1932	51	...	2	12	37
1933	5	1	2	2	...
1934	10	...	6	...	4
1935	22	...	10	5	7
1936	35	1	8	15	11
1937	26	...	2	16	8
1938	19	2	6	4	7
1939	23	1	4	11	7
1940	34	1	7	21	5

All the fatal cases were reported to the Bureau of Vital Statistics. However, there is little tendency to report nonfatal lead poisoning. Lead poisoning in children is not a reportable disease in Maryland. Since 1934 at least 117 cases of nonfatal lead poisoning have occurred in all ages, yet 89 per cent of these cases were ascertained only as a result of a follow-up study of the blood lead laboratory service. The table includes 40 cases of nonfatal lead poisoning which occurred in 1932 as a result of the use of discarded storage battery casings for fuel.[2] Fatal lead poisoning was far more frequent happening in children than in adults. Eighty-six per cent of the deaths recorded in table 1 were those of children, the primary cause of death in practically all being either lead poisoning or lead encephalitis. On the other hand, in 5 of the 7 adult deaths lead poisoning was reported merely as a contributory cause.

The high frequency of fatal lead poisoning in children reported from Baltimore as compared with other areas

1. Kaplan, Emanuel, and McDonald, J. M.: Blood Lead Determinations as a Health Department Laboratory Service, Am. J. Pub. Health **32**: 481 (May) 1942.

2. Williams, Huntington; Schulze, W. H.; Rothchild, H. B.; Brown, A. S., and Smith, F. R., Jr.: Lead Poisoning from the Burning of Battery Casings, J. A. M. A. **100**: 1485 (May 13) 1933.

is evident in table 2, compiled from information furnished by the United States Bureau of Census. Of the 202 deaths from lead poisoning in persons under 15 years of age which were reported in the entire United States registration area (1936 population about 128,052,000), 49 deaths, or 24.3 per cent of the total, were from the city of Baltimore (1936 population about 838,700), which constitutes only 0.65 per cent of the population of the United States registration area. Based on cases reported in children during the ten year period 1931-1940, the death rate from lead poisoning in Baltimore would seem to have been about fifty times as high as in the remainder of the general population of the United States. That this situation was not generally appreciated is evident from the following statement, which appeared in a statistical report [3] on lead poisoning:

> A tabulation of deaths from lead poisoning in eighteen American cities in which lead-using industries are more or less concentrated gives the following results: In the aggregate there were 179 deaths during the period 1929-36, of which 30 occurred in New York City, 29 in Baltimore, 28 in Philadelphia, 22 in Boston, 16 in Chicago, 15 in Cleveland and 10 in Providence.

This statement implied that the relatively large number of deaths from lead poisoning in Baltimore had occurred as a result of industrial exposures. However, the implication was not in accordance with the known facts. Actually 36 cases of fatal lead poisoning were reported in Baltimore during the period 1929-1936. Only 4 of these deaths occurred in adults. On the other hand, 32 deaths, or 89 per cent of the total, were due to nonindustrial lead poisoning; that is to say, they were deaths of children, mostly resulting from the chewing of objects painted with lead-containing paint. These facts again demonstrate the general lack of adequate statistical information on mortality and morbidity from lead poisoning, a condition which has been repeatedly mentioned in the literature.[4]

TABLE 2.—*Number of Reported Deaths from Lead Poisoning by Age Groups in Selected States and in the United States, 1931-1940*

	All Ages	Under 15 Years	15 Years and Over
Total United States registration area	1,055	202	853
California	51	2	49
Illinois	70	6	64
Maryland	60	49	11
City of Baltimore *	56	49	7
Massachusetts	64	31	33
Michigan	30	3	27
Missouri	33	1	32
New Jersey	56	10	46
New York	142	36	106
Ohio	81	5	76
Pennsylvania	83	18	65
Remainder of United States registration area	385	41	344

* The population of the city of Baltimore is about 47 per cent of the total population of the state of Maryland.

TABLE 3.—*Lead Poisoning in Children in Baltimore, 1931-1940*

Age, Years	Total	White		Colored	
		Male	Female	Male	Female
Total	135	25	19	46	45
Under 1	1	..	..	1	..
1	26	6	7	8	5
2	45	15	7	10	13
3	23	..	3	4	6
4	10	3	1	2	4
5-9	27	1	1	15	10
10-14	13	..	..	6	7

During the period 1931-1940 a total of 135 cases of lead poisoning occurred in children in Baltimore City. The age, sex and color distribution of these children is shown in table 3, which includes both fatal and nonfatal cases. The average age in this group is 4 years. However, the group includes 37 colored children of an average age of 7½ years who were concerned in the 1932 outbreak of lead poisoning associated with the use of storage battery casings for fuel.[2] The average age of the remaining 99 children is 2½ years. Practically all had a history of pica associated with the chewing of objects painted with lead-containing paints. The age specific morbidity rate for lead poisoning during the ten years 1931-1940 based on this group of 99 children is more than five times as high among the colored population (13.7 per hundred thousand) as it is among the white population (2.5 per hundred thousand). The colored group constitutes approximately 19 per cent of the total population.

During the same time 93 cases of lead poisoning occurred among adults. Colored persons were involved in 14 of these cases. The age specific morbidity rate among colored adults over the ten year period (1.2 per hundred thousand) is less than the rate among the white persons (1.6 per hundred thousand). For the five years 1936-1941, which reflect the increase in our knowledge of the occurrence of lead poisoning (table 1), the corresponding morbidity rates in adults are somewhat higher (1.5 per hundred thousand in the colored group and 2.5 per hundred thousand in the white group).

TABLE 4.—*Occupational Distribution of Persons with Lead Poisoning in Baltimore, 1931-1940*

Occupation	Number of Cases		
	Total	White	Colored
Total	228	123	105
Painter	24	22	2
Lead arsenate worker	13	13	..
Acetylene burner	12	11	1
Enamel worker	10	10	..
Paint maker	8	5	3
Gasoline tank cleaner	8	8	..
Junk metal worker	5	1	4
Solder grinder	3	3	..
Printer	1	1	..
Miscellaneous occupations	5	4	1
Nonoccupational, adult	4	1	3
Nonoccupational, children	135	44	91

An occupational distribution of persons with lead poisoning is shown in table 4. The largest single group includes painters. It must be pointed out, however, that a number of painters in this series had relatively mild lead intoxication. Nine of the 13 lead arsenate workers were white women employed in packing operations. One of the enamel workers was a white woman engaged in spraying enamel frit. There were no other cases of lead poisoning of occupational origin among women. The junk metal workers operated furnaces for reclaiming scrap lead in junk yards. The acetylene burners employed torches in shipbreaking. The printer

3. Hoffman, F. L.: Lead Poisoning in 1936 and in Earlier Years, Monthly Labor Review, Bureau of Labor Statistics, U. S. Department of Labor, February 1938, Serial No. K. 669.

4. Lanza, A. J.: Epidemiology of Lead Poisoning, J. A. M. A. 104: 85 (Jan. 12) 1935. Hamilton, Alice: The Prevalence of Industrial Lead Poisoning in the United States, chapter XX in Lead Poisoning, by J. C. Aub, L. T. Fairhall, A. S. Minot and Paul Reznikoff, Medical Monographs, vol. 7, Baltimore, Williams & Wilkins Company, 1926. McCord, C. P., and others: Lead Poisoning in the United States, Am. J. Pub. Health 19: 631 (June) 1929.

spent a portion of his daily time remelting type and skimming and sifting dross from the remelt pot. The list includes one nonoccupational case, that of an adult who was given lead and opium pills in the treatment of tuberculous enteritis.[5]

SUMMARY

1. Largely as a result of studies associated with a follow-up of a blood lead laboratory service, it has been possible for the Baltimore City Health Department to acquire relatively accurate information concerning the incidence of lead poisoning in the community in recent years.

2. In the ten year period 1931-1940 at least 228 cases of lead poisoning are known to have occurred in Baltimore. Of these, 93 cases, or 41 per cent, involved adults, and 135 cases, or 59 per cent, occurred in children. There were 7 fatalities among the adults, as compared with 49 fatalities among the children.

3. Lead poisoning was of more frequent occurrence among colored children than among white children. Adult lead poisoning of occupational origin was more common among the white population than among the colored.

ECLAMPSIA AT THE UNIVERSITY HOSPITAL

1926 TO 1941

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During the sixteen years between Jan. 1, 1926 and Dec. 31, 1941, 80 eclamptic patients were observed and treated at the University Hospital. Thirty-seven women were admitted in the convulsive stage, and 43 suffered the seizures after admission; all but 13 of the latter were admitted because of toxemia. Throughout the series, eclampsia was viewed as a medical condition and treatment followed a fairly consistent pattern. This communication deals with the results obtained and involves a discussion of the lessons learned by the experience.

TREATMENT EMPLOYED

The essence of therapy revolves around the clinical observation that eclamptics generally do not have convulsions when the respirations are less than 14 to 16 per minute and the deduction therefrom that there is some relationship between the respiratory exchange and the appearance of the convulsive episodes. Heavy sedation was therefore employed, and the dosage was regulated solely by the effect on the respiratory rate, which was reduced to 12 per minute as quickly as possible. The most commonly employed drug was morphine, which was administered in all but 6 very mild cases in amounts varying from ¼ grain (0.016 Gm.) to 6⅔ grains (0.44 Gm.), with the average approximating 1¼ to 1½ grains (0.08 to 0.1 Gm.). Chloral hydrate (53 cases), magnesium sulfate parenterally (28 cases) and various barbiturates (15 cases) were employed to complement the morphine; these drugs are all respiratory depressants.

Other forms of therapy employed included venesection in 18 cases, mostly in the earlier years, spinal tap in 2, oxygen in 11 (7 during 1941), concentrated dextrose solutions intravenously in 49 and concentrated sucrose solutions in 10, mostly since 1932. Scopolamine hydrobromide, atropine, theophylline with ethylene diamine and mercuropurin were each given to 1 to 3 patients on special indication. Transfusions were employed twice in the presence of shock and hemorrhage, but both patients died.

MATERNAL DEATHS

The total maternal deaths numbered 7, a fatality rate of 8.75 per cent. Six of the 7 women who died had antepartum (4) or intrapartum eclampsia (2) before admission, while the other was a 45 year old primigravida who was delivered spontaneously, had a single convulsion one hour and twenty minutes later and died from uncontrollable uterine hemorrhage and shock (fibroid uterus) four hours after the seizure. Autopsy was permitted for 3 of the other 6 patients and showed the deaths to be due to (1) infection (Escherichia coli septicemia) after vaginal hysterotomy, (2) bilateral pneumonia with infarction of the anterior lobe of the hypophysis (normal liver) and (3) cardiac dilatation, pulmonary edema and eclamptic liver. Two of the patients for whom necropsy was not permitted died of shock (they had been brought 30 and 150 miles respectively to the hospital while having frequent convulsions), while the third had clinical failure of the left side of the heart and pulmonary edema unrelieved by venesection.

FETAL SURVIVAL

The survival rate among the 84 infants (4 sets of twins) was 53.6 per cent (45 lived), with the salvage according to birth weights as indicated in the accompanying table.

Exclusion of the 18 "previable" children, weighing less than 1,500 Gm., among whom there was only 1 survivor, leaves 66 with a reasonable chance of survival. The actual salvage was 44, or 66.7 per cent. Three of the other 22 children died during the first day, while 19 were stillborn, including 9 that were macerated. The toxemia appears, therefore, to have been the direct cause of the high fetal death rate either by inducing or necessitating premature delivery or by producing intrauterine fetal death.

INCIDENCE

During the interval under consideration there were 15,327 deliveries, and the incidence of eclampsia was 1 in 191. The yearly incidence varied from 1 in 57 deliveries in 1927 to 1 in 956 in 1940, for no determinable reason. The greatest numbers of cases occurred in May (11) and June (10) and the smallest in August (2). There were 52 in the first six months as against 28 in the second half of the calendar years. This distribution supports the old clinical observation that the late spring months show an increased eclamptic incidence. Three of the fatalities occurred in the first six months (3 in 52, or 1:17), while there were 4 in the second six months (4 in 28, or 1:7). This suggests that eclampsia may be less severe when it is more frequent.

PARITY

Primigravidity is recognized as predisposing to eclampsia, and 59 women (73.7 per cent) of this series had not previously been pregnant. Greater interest, however, attaches to the 21 multigravidas in whom convulsions developed. The number of previous pregnancies varied from one to twelve, and 13 women had had three or more. According to the histories, 12 had not had toxemia previously, although the majority had had.

5. Geraghty, W. R.: Encephalopathy from the Therapeutic Use of Lead and Opium Pills, J. A. M. A. 110: 208 (Jan. 15) 1938.

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