The following method was used. A small enema was given to constipated children a few hours before the proposed administration, to help the absorption of the drug and minimize the risk of its return after injection. No food was given for at least three hours before the thiopentone was due. A hypodermic injection of atropine, 1/150 or 1/100 gr. (0.43 or 0.65 mg.), was given half an hour before the thiopentone in almost all cases. This is not essential, but is useful in controlling salivation.

The thiopentone was dissolved in 20 to 40 ml. of warm tap-water and injected with a syringe slowly into the rectum by a rubber catheter. This injection took at least five minutes to complete. Afterwards the buttocks were held or strapped together to prevent some of the drug being returned. When this was not prevented, half the original dose was given half an hour later, if the child was not sufficiently sedated. The dose employed was 1 g. per 50 lb. of body weight. A detailed schedule is given in the accompanying Table.

Dosage Schedule of Thiopentone for Children Weighing up to 52½ lb.

Weight of Child		Dose of Thiopentone
lb.	kg.	
7.5-12.5	(3.40-5.67)	0.2 g.
12.6-17.5	(5.71-7.93)	0.3 g.
17.6-22.5	(7.98-10.20)	0.4 g.
22.6-27.5	(10.25-12.47)	0.5 g.
27.6-32.5	(12.51-14.74)	0.6 g.
32.6-37.5	(14.79-17.01)	0.7 g.
37.6-42.5	(17.06-19.28)	0.8 g.
42.6-47.5	(19.32-21.54)	0.9 g.
47.6-52.5	(21.59-23.81)	1.0 g.

The child was usually asleep by the time the injection was completed, and then was left undisturbed lying on its side for at least another five minutes to ensure adequate sedation.

When examination of the fundi was planned the pupils were previously dilated with 1% homatropine drops.

Results

With two exceptions the results were uniformly satisfactory. Systematic examination of the eyegrounds, with a detailed description of all abnormalities and their precise location, could be completed in five minutes. As the desired effect of the thiopentone lasted for nearly an hour in all cases, it was possible for several observers to complete the examination or to demonstrate the findings to a class of students without tiring the patient. There was just enough time to complete an encephalographic examination. Although the children were sufficiently awake to take meals after some three hours they usually slept most of the next 24 hours. This was of great benefit in diminishing headache following encephalography. There was complete amnesia for 24 hours even if the child reacted to a painful stimulus, such as lumbar puncture.

In a few cases cystoscopy or laryngoscopy was performed successfully without any additional sedation, but if any surgical incision was needed—as, for example, the opening of an abscess or the making of cranial burr-holes—local analgesia had to be used as well.

In the absence of painful stimuli complete muscular relaxation was obtained. This was useful for abdominal palpation, usually carried out in addition to the original purpose of the sedation. An enlarged spleen or liver was easily felt. This method could therefore be used in particularly difficult cases for the diagnosis of abdominal disorders, though I have had no personal experience of rectal thiopentone given specifically for this purpose.

In no case was any general or local, immediate or remote toxic effect observed. Nevertheless, it is advisable to have

oxygen and an injection of nikethamide or picrotoxin in readiness in case of excessive sedation.

One infant was in danger of dying after aspirating stomach contents, but made a complete recovery. Another child had severe spasms of coughing with temporary cyanosis. These two accidents were due to failure to observe the correct technique: one was fed just before thiopentone was given, and the other did not receive atropine.

Rectal thiopentone should not be given within three hours of a meal. It is contraindicated in the presence of respiratory obstruction, painful conditions of the rectum, or diarrhoea. Otherwise it is safe even for extremely ill and semiconscious children. It is an excellent method for ophthalmoscopic examination of babies.

Summary

Rectal thiopentone was given on 113 consecutive occasions to 64 children aged from 3 months to 12 years. The dose was 1 g. per 50 lb. of body weight. Complete and satisfactory sedation was obtained for examination of the fundus oculi, encephalography, and other procedures not requiring surgical incision.

No local or general toxic effects were observed.

Detailed technical instructions are given.

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CONVULSIONS IN CHILDHOOD AN EPIDEMIOLOGICAL NOTE ON THEIR ASSOCIATION WITH EPILEPSY, ECLAMPSIA, AND STILLBIRTH

BY

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Nathan and Ransford (1949) have adduced evidence in favour of the claim that convulsions in infancy are the early manifestation of epilepsy, and Grantly Read (1950) has drawn attention to the possible relationship which they may have to certain types of convulsion in pregnancy and labour. Since the commonest cause of convulsions in pregnancy and labour is eclampsia, which is not infrequently associated clinically with stillbirth, it is of interest to note that certain epidemiological kinships exist between infantile convulsions and epilepsy, eclampsia, and stillbirth. These kinships may be considered under (1) secular trend, (2) seasonal variation, and (3) regional distribution, limiting the investigation to mortality data relating to England and Wales.

Secular Trend

Mortality from these diseases has been declining for many years, and the latest available figures show that unprecedentedly low rates now obtain. Mortality from convulsions in infants under 1 year has fallen from 9.68 per 1,000 births in 1911 to 0.35 in 1947, and deaths at all ages from epilepsy show a decline—interrupted, however, during the two world wars—from 72 per million in 1911 to 29 in 1947. Mortality from eclampsia has fallen sharply since

its peak in 1919, and stillbirths, which became registrable in 1927, have declined since 1935. It may be noted that neonatal mortality, which has close ties with stillbirth, has declined from 41 per 1,000 births in 1911 to 23 in 1947. This declining trend, which is in common with that given by many other diseases, does not impart distinction to the group, though it deserves mention as a feature of the individual components of it.

Seasonal Variation

It will be noted (Table I) that in the years shown the highest number of deaths from infantile convulsions and epilepsy occur during January and February, and the

TABLE I.—*Seasonal Distribution of Deaths from Infantile Convulsions (Under 5 Years of Age), Epilepsy, Eclampsia, and of Stillbirths, Corrected for Difference in Length of Months, in the Years 1939–45, in England and Wales*

	Deaths			
	Infantile Convulsions	Epilepsy	Eclampsia	Stillbirth
January ..	582 (12%)	1,151 (10%)	241 (9%)	13,663 (9%)
February ..	557 (11%)	1,151 (10%)	237 (9%)	13,760 (9%)
March ..	502 (10%)	1,099 (10%)	234 (9%)	14,098 (9%)
April ..	481 (10%)	989 (9%)	256 (9%)	13,698 (9%)
May ..	374 (8%)	939 (8%)	249 (9%)	13,476 (9%)
June ..	338 (7%)	895 (8%)	230 (9%)	12,782 (8%)
July ..	326 (7%)	792 (7%)	228 (9%)	12,556 (8%)
August ..	270 (6%)	768 (7%)	215 (8%)	12,092 (8%)
September ..	302 (6%)	799 (7%)	202 (7%)	12,170 (8%)
October ..	323 (7%)	793 (7%)	209 (7%)	12,234 (8%)
November ..	374 (8%)	888 (8%)	181 (7%)	11,958 (8%)
December ..	440 (9%)	978 (9%)	228 (9%)	12,647 (8%)
Total ..	4,869	11,242	2,710	155,134

lowest in August. The curves, in fact, are fairly parallel though the amplitude is less in the case of epilepsy. Deaths from eclampsia are above the average from January until July, and stillbirths from January until May, and both fall to a minimum in November. The seasonal variation of mortality from eclampsia is roughly similar to the curve given by stillbirth. It should be added here that, in the Tables in this study, deaths assigned to the toxæmias of pregnancy and to the puerperal toxæmias have been included in the estimation of mortality from eclampsia as well as deaths associated with convulsions.

Regional Distribution

The regional variations in mortality are closely similar (Table II), a feature which is revealed by the correlations between the rates. The coefficients are all of the order +0.8 or +0.9, and are highly significant in the statistical sense. The greatest coefficient is given by epilepsy and stillbirth in the triennium 1945–7 ($r = +0.93$), but its importance is diminished by the fact that the epilepsy rate is not adjusted to the differing age and sex constitution of the regions. This defect, however, is less than might be expected, because deaths from epilepsy are not heaped up in one or two age groups, but fairly well spread throughout all, and, moreover, the regional variations shown are considerable.

The stillbirth rates used in this study, it should also be noted, are not corrected for differences in biological factors such as age and parity of mother, but it is known that these do not influence the regional variations to more than a relatively slight extent. The coefficient between stillbirth and mortality from infantile convulsions in the 1932–7 period is +0.87. The coefficient relating to the same heads in 1939–46 is reduced to +0.78, but is still highly significant. The reduction is possibly due to changes caused by evacuation in the war years. In the correlation between puerperal toxæmia and infantile convulsions during 1939–46, $r = +0.80$. The interesting point about these coefficients

TABLE II.—*Regional Stillbirth Rates and Regional Mortality from Infantile Convulsions, Epilepsy, and Eclampsia. NOTE: For the Geographical Constitution of the Regions See the Registrar-General's Review, Any Recent Issue*

Region	Average Annual Stillbirth Rate per 1,000 Live Births and Stillbirths			Convulsions. Proportion of Deaths Under 1 Year to 1,000 Births		Epilepsy. Crude Death Rate per Million Persons Living, 1945–7	Average Annual Death Rate from Eclampsia per 1,000 Live Births and Stillbirths, 1939–46
	1932–7	1939–46	1945–7	1932–7	1939–46		
	1	2	3	4	5	6	7
Greater London ..	32	27	23	0.42	0.19	73	0.42
Remainder of S.E. ..	35	28	24	0.84	0.35	79	0.54
North I ..	42	35	29	3.75	2.55	110	0.64
North II ..	42	33	29	2.39	1.10	98	0.60
North III ..	46	35	28	1.98	0.88	86	0.55
North IV ..	46	36	29	2.11	1.19	107	0.55
Midland I ..	40	31	26	1.29	0.56	92	0.56
Midland II ..	41	31	26	2.08	1.07	89	0.55
East ..	38	31	26	1.34	0.57	103	0.52
South-west ..	40	31	25	1.31	0.50	89	0.68
Wales I ..	55	40	32	4.36	2.67	134	0.77
Wales II ..	52	38	31	4.40	2.65	121	0.82

$r_{14} = +0.87$, $r_{16} = +0.89$, $r_{25} = +0.78$, $r_{26} = +0.88$, $r_{27} = +0.77$, $r_{36} = +0.93$, $r_{46} = +0.89$, $r_{56} = +0.93$, $r_{57} = +0.80$, $r_{67} = +0.83$. Note: In each case the first two numbers refer to columns—e.g. r_{14} means coefficient of correlation of columns 1 and 4.

is that they reveal, within the limits of the regional basis, statistical relationships between infantile convulsions, epilepsy, eclampsia, and stillbirth which are at least as important as the relationship shown between eclampsia and stillbirth, where $r = +0.77$. And, as this last relationship coincides with the known clinical association of eclampsia and stillbirth, it seems natural to ask if the other relationships may not also indicate equally important aetiological factors in common.

A further point of correspondence between epilepsy and infantile convulsions is that they both show an excess of male mortality—epilepsy at all ages (Robertson, 1949). In this they resemble stillbirth and neonatal mortality.

The evidence given in this note suggests that convulsions in childhood may be related to convulsions in pregnancy and labour and that they are the early manifestation of epilepsy. The correspondences suggest that common factors are at work, one of which may be the distribution of the incidence of infectious diseases. Further investigation of this wider field, which now includes stillbirth, is justifiable.

Summary

Attention is drawn to some epidemiological kinships between infantile convulsions, epilepsy, eclampsia, and stillbirth.

Mortality trends have been falling for many years, with the possible exception of stillbirth, for which the rate was not recorded until 1927. The neonatal rate shows a corresponding decline.

The seasonal curves of mortality from infantile convulsions and epilepsy are similar, and deaths from eclampsia show a rough correspondence with stillbirths, though the seasonal variation of stillbirths is not so great. In all cases mortality is highest in the first half of the year.

A close correspondence is noted in the regional distributions of mortality, and reference is made to the excess of male deaths in infantile convulsions and epilepsy and in foetal and neonatal mortality.

The seasonal distribution of stillbirths and the rates relating to eclampsia and epilepsy were calculated from data kindly supplied by the Registrar-General. The other rates were calculated from data published in the Registrar-General's Reviews.

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