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OPHTHALMIA NEONATORUM



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OPHTHALMIA
NEONATORUM

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THE PROBLEM AFTER THIRTY YEARS OF STATUTORY
NOTIFICATION AND SIXTY YEARS OF CREDÉ PROPHYLAXIS

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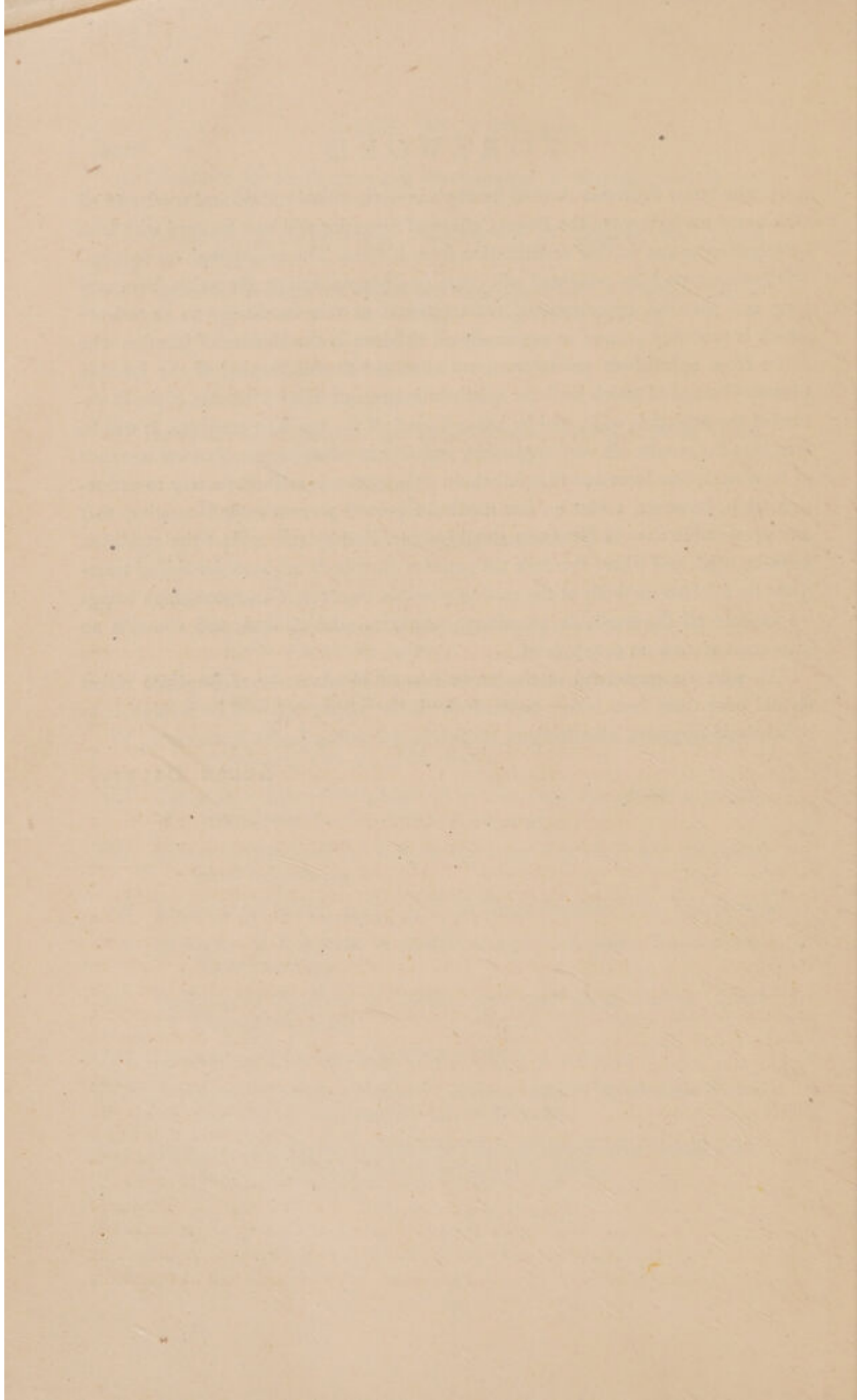
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FOREWORD

ALL who know Professor Arnold Sorsby's investigational and clinical work and all who heard his lectures to the Royal College of Surgeons will look forward with keen anticipation to any further contribution from his pen. The monograph on ophthalmia neonatorum here presented will cause no disappointment. He writes from very long and personal experience of the treatment of this condition, an experience which is probably unique, as practically all children in the County of London who suffer from ophthalmia neonatorum are admitted to the hospital of the London County Council of which he is the ophthalmic surgeon. His knowledge of the literature of the subject is wide, and he has collected all the available statistics. It will be seen that the results are very creditable, and that in consequence of newer methods of treatment, blindness due to ophthalmia neonatorum is well on its way to extinction. It is, however, a blot on our medical services, preventive and curative, that any preventible case of blindness should occur. Prophylaxis against the condition must be used, and where this fails the patients must be brought under skilled treatment by modern methods at the earliest possible moment. This monograph brings up to date all the available knowledge, statistical and clinical, and contains an important section on prophylaxis.

All who are concerned in the prevention of blindness or of impaired vision should have these facts before them. Indeed, the monograph will be invaluable to ophthalmic surgeons, obstetricians, midwives and public health workers.

ALLEN DALEY.



STATISTICAL

Ophthalmia Neonatorum does not appear to be prevalent universally. It is a curiosity in Egypt, where the habit of native midwives to put either a mixture of onion-juice and salt or the juice of a lemon and occasionally various native preparations into the eyes of new-born babies has been advanced as a possible cause for the rarity of the affection (Wilson, 1939). This rarity is all the more striking as epidemics of gonococcal ophthalmia amongst older children and adults are distressingly frequent and constitute the major cause of blindness in this classical country of the blind. In Palestine the position is similar; the adult peasant population is racked with acute ophthalmia, which, as in Egypt, is the principal cause of blindness and is frequently gonococcal in origin, but ophthalmia neonatorum is negligible (Shimkin, 1928; Strathearn, 1933). In Tunisia a somewhat similar state of affairs holds good (Toulant, 1936).

That some factor other than geographical position is operative in the rarity of the affection in these adjacent Mediterranean countries is suggested by the infrequency of the disease in Soviet Mongolia. In this primitive area gonorrhoea is widely prevalent (estimated as high as 24% in the population), blindness is very common—as much as 4.8% of the population being afflicted—but ophthalmia neonatorum is rare, and runs a mild course when it does occur (Geulikman, 1939; Geulikman and Zalutski, 1940). In China ophthalmia neonatorum figures at 0.2%, and gonorrhoeal ophthalmia in adults as 3.6% in the causes of blindness reported by Ling (1923). Other statistical data (Ling, 1923; Chang, 1930; Cunningham, 1936) also seem to point to ophthalmia neonatorum as an infrequent cause of blindness, but this may be a purely relative state of affairs in view of the heavy incidence of blindness from other causes. In India it appears to be infrequent and to run a mild course (Wright, 1931, 1934; Bagchi, 1935). It is also infrequent in Dutch East India (Houwer, 1939).

In most other countries ophthalmia neonatorum is a formidable problem. In the South American republics it is still one of the major, if not the commonest cause, of blindness, being responsible for some 25–48% of all blindness seen at institutes for the blind in Argentina, Brazil, Columbia and Uruguay (Vasquez-Barrière, 1939). In the U.S.A. it constitutes an important though dwindling cause of blindness. In a series of 4,604 blind children studied by Kerby in 1941–42, 10.5% were blind from this affection. That ophthalmia neonatorum is widespread is seen from the fact that a special investigation of the records of New York City hospitals revealed an incidence of 7 per 1,000 births in 1931–36 (Lowenstein). More recently Blumberg and Gleich (1943) reported that 213 cases of gonococcal ophthalmia neonatorum had been notified in New York during 1938–42.

It is difficult to assess the frequency of ophthalmia neonatorum on the European continent. Though the disease is notifiable in France and Switzerland, and under certain conditions in Italy, notification returns are scanty. The system of notification in force in England and Wales since 1914 is only equalled by that obtaining in Scotland. Prior to 1914 the affection had been notifiable in the areas of several local authorities, but a General Order issued in that year made notification compulsory throughout England and Wales. For the purpose of this Order the affection was defined as a purulent discharge from the eyes of an infant commencing within 21 days from the date of its birth. The Order obliged midwives and medical attendants

to notify the disease, and local authorities to provide treatment or to satisfy themselves that it was available. Since 1936 the duty of notification has devolved entirely on the medical attendant, that of midwives being covered by the rule of the Central Midwives Board which requires a midwife to summon medical assistance for a case of "any inflammation or discharge from the eyes, however slight".

Notifications are made to the Local Health Authority which in turn transmits them both to the County Health Authority and to the Registrar-General. At the end of each year the Local Health Authorities submit to the Ministry of Health a return showing the fate of the notified cases. The returns given by the Registrar-General in his *Annual Statistical Review*, together with data obtained from the Ministry of Health, blind schools and hospitals, form a substantial statistical basis for a study on the incidence and significance of ophthalmia neonatorum as a cause of impaired vision and blindness in England and Wales.

I. THE INCIDENCE OF OPHTHALMIA NEONATORUM IN ENGLAND AND WALES

THE first full year of notifications in England and Wales (1915) showed an incidence of ophthalmia neonatorum of 8.34 per 1,000 births, and a somewhat similar rate obtained during 1923-31, since when it has tended to decline, the rate for 1943 being 6.59 per 1,000. The years 1916-22 showed a rate between 9 to over 12 per 1,000, the peak year being 1919 with a rate of 12.49 per 1,000 births (Table I).

A distinctly wide difference in rates of notifications in different parts of the country is covered by these figures. This is seen from Table II, which sets out the rates separately for Administrative Counties, County Boroughs and Metropolitan Boroughs contrasted with the rate for the whole of England and Wales. Generally speaking, the Metropolitan Boroughs show an incidence of the same order as the general rate; the County Boroughs show a rate almost double, and the Administrative Counties one of almost half the general rate. These seemingly consistent trends are, however, not borne out by an analysis of the rates for individual county boroughs, and four extreme examples are shown in the table (Table II). Birmingham and Leeds showed in 1918 fairly similar rates somewhat above the general rate (13.3, 11.8 and 9.9 per 1,000 respectively); but whilst in Birmingham the rate has been rising steeply to reach 63.5 per 1,000 in 1938, the rate for Leeds has shown a steady decline to 4.2 per 1,000 in 1938. The rate for Birmingham has increased by almost 5 times, and that for Leeds has fallen to less than half; or to put it otherwise, two large county boroughs which in 1918 showed a fairly similar rate, somewhat higher than the general rate, show 21 years later an incidence in the one case almost 8 times the incidence for the country as a whole and in the other an incidence almost half that of the country. Equally striking are the experiences recorded for Liverpool and Manchester. Both these cities showed in 1918 a distinctly higher rate than that for the country as a whole (34.4, 30.1 and 9.9 per 1,000 respectively), but whilst the high rate for Liverpool has remained relatively stationary except for a rise in recent years (1934-38), that for Manchester has tended to decline to a level approaching the general rate.

These rates are, of course, based on individual returns, and there is evidence that local variations in the interpretation of the Order for compulsory notification rather than actual differences in incidence are responsible for the vagaries the statistics display. This is confirmed by the replies received to a questionnaire addressed to a number of the more important lying-in institutes. Information was

sought on the total number of cases of ophthalmia neonatorum as a whole and of gonococcal ophthalmia in particular that occurred at these various centres during 1938-42. The following table summarizes some typical replies.

Number of births at the particular institution during the period 1938-42	Number of cases of Ophthalmia Neonatorum	Number of cases of Gonococcal Ophthalmia Neonatorum
12,149	1,216	15
9,790	25	19
9,119	210	10
7,479	25	8
2,398	51	5
1,050	0 (but 38 cases of "sticky eye" not regarded as notifiable were observed)	0

In the light of these observations it would appear that the relatively consistent rates recorded by the Registrar-General cannot be taken as an unquestionable indication of the incidence of ophthalmia neonatorum and that the variations they show from year to year and for different parts of the country have to be assessed with reserve. Notification primarily serves, of course, the purpose of ensuring early and adequate treatment and not that of collecting statistics. It is therefore of little moment if the returns include categories of ophthalmia that carry no danger to sight; the purpose of notification is satisfied if some cases of infantile ophthalmia that might have ended in the disaster of blindness had, as a result of the statutory enactment, been provided with adequate treatment. For this reason a study of the incidence of visual impairment and blindness from ophthalmia neonatorum over some years is relevant.

2. THE INCIDENCE OF IMPAIRED VISION AND OF BLINDNESS CONSEQUENT ON OPHTHALMIA NEONATORUM

IN 1921 there were 82 cases of impaired vision and 7 of blindness amongst 4,555 cases of ophthalmia neonatorum notified by 83 Local Health Authorities (Ministry of Health, 1922).

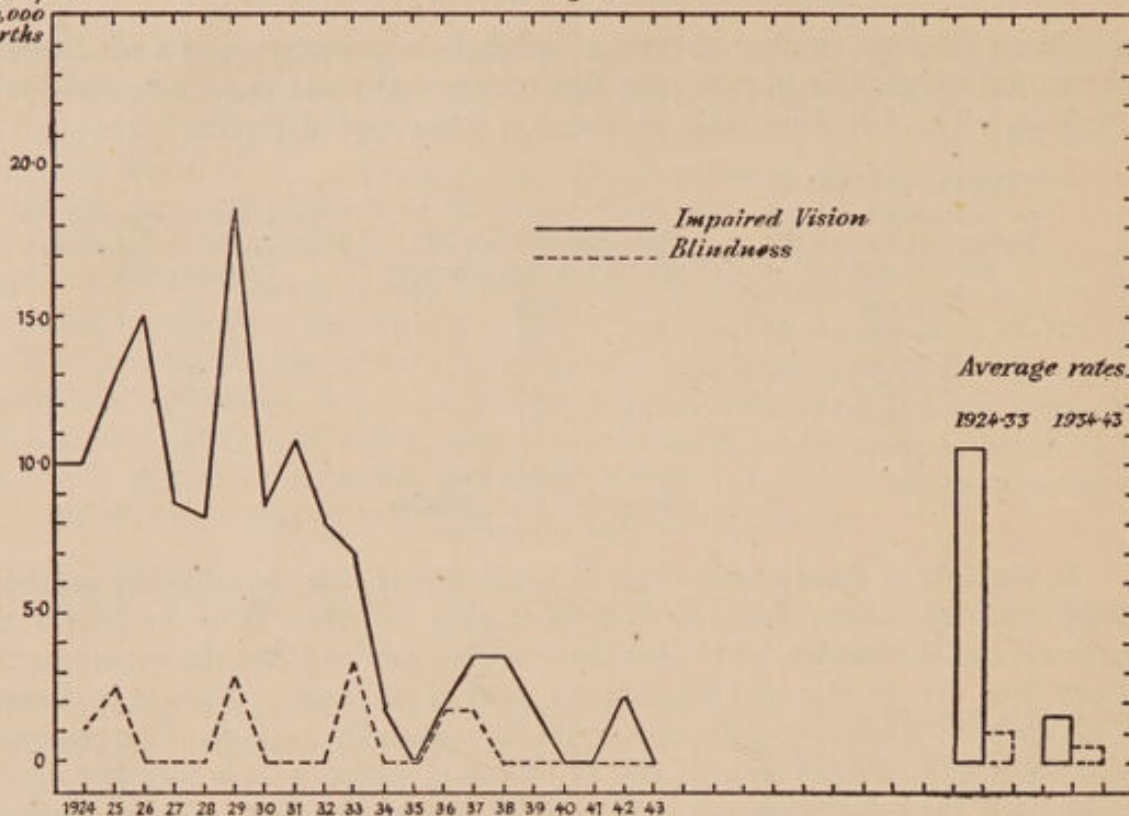
For the period 1924-33 returns from the Metropolitan Boroughs and from the County Boroughs of England and Wales (with a few exceptions) are available. They are set out in Table III. Available comparative data for 1934-43 are included in the returns for the whole country (Table IV), and the following summary table brings out a striking decline in the incidence of impaired vision and of blindness from ophthalmia neonatorum. (See also Figs. 1 and 2.)

Metropolitan and English County Boroughs, 1924-43

	Notifications	Vision Impaired		Blind	
		No.	Rate per 100,000 births	No.	Rate per 100,000 births
<i>Metropolitan Boroughs</i>					
1924-33	6,898	76	10.7	7	1.0
1934-43	3,613	8	1.6	7	0.4
<i>County Boroughs (England)</i>					
1924-33	27,566	394	18.7	46	2.2
1934-43	21,901	90	4.5	18	0.9

Rate per
100,000
births

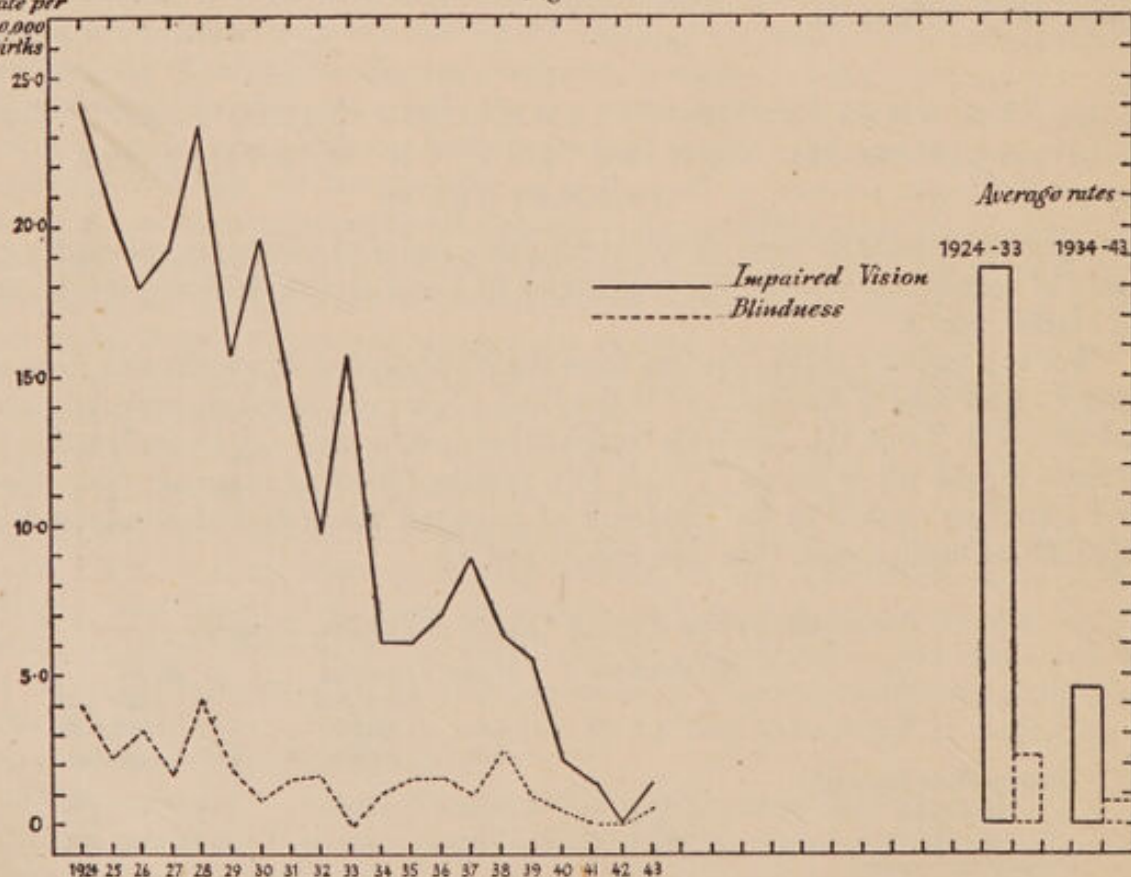
Fig.1.



Impaired Vision and Blindness from Ophthalmia Neonatorum.
Annual rates for London County Council (Metropolitan Boroughs): 1924-43.

Rate per
100,000
births

Fig.2.

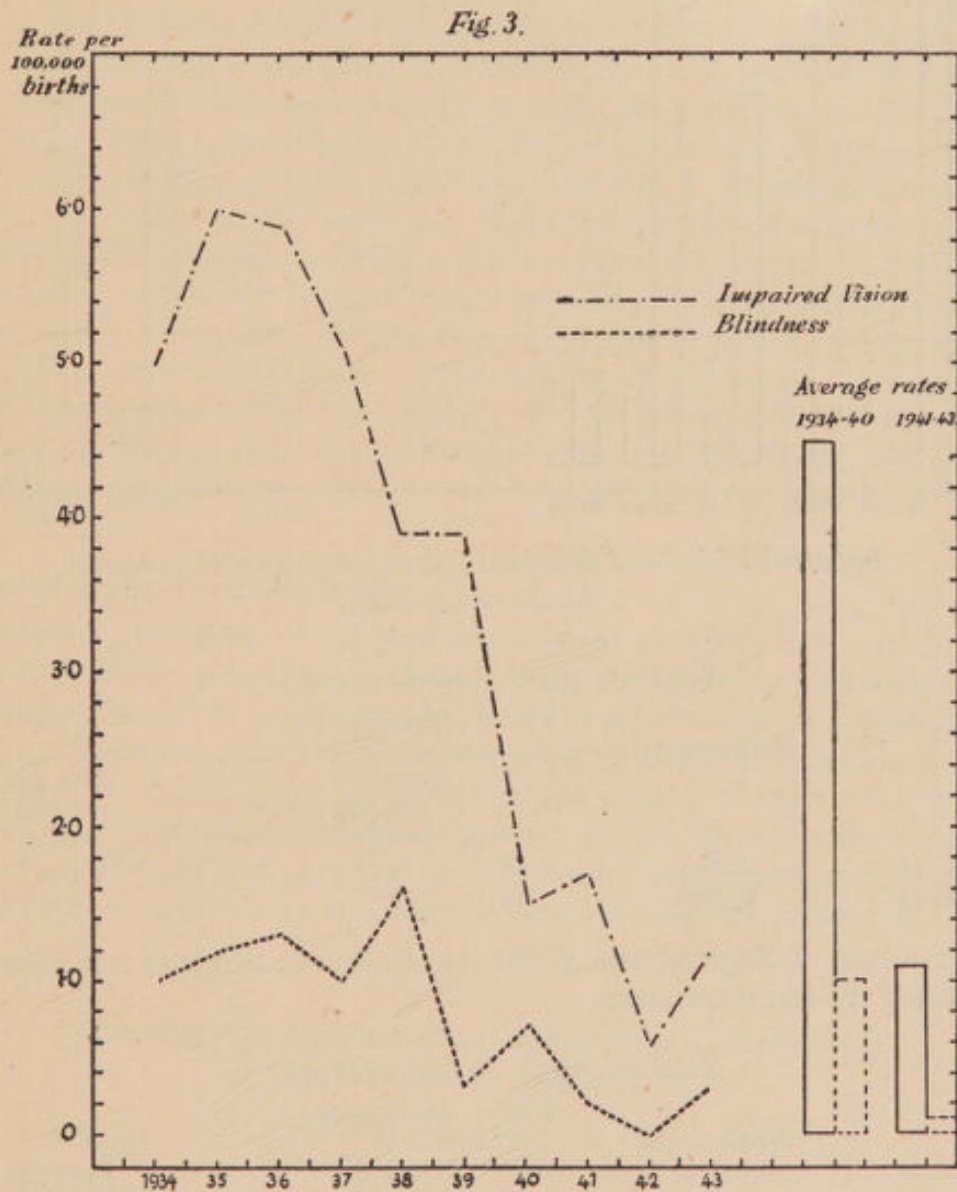


Impaired Vision and Blindness from Ophthalmia Neonatorum.
Annual rates for County Boroughs (England): 1924-43.

The decline has been particularly marked during the last few years (Fig. 3). This is brought out by comparing the rates for 1941-43 with those for 1934-40 (Table IV (6)).

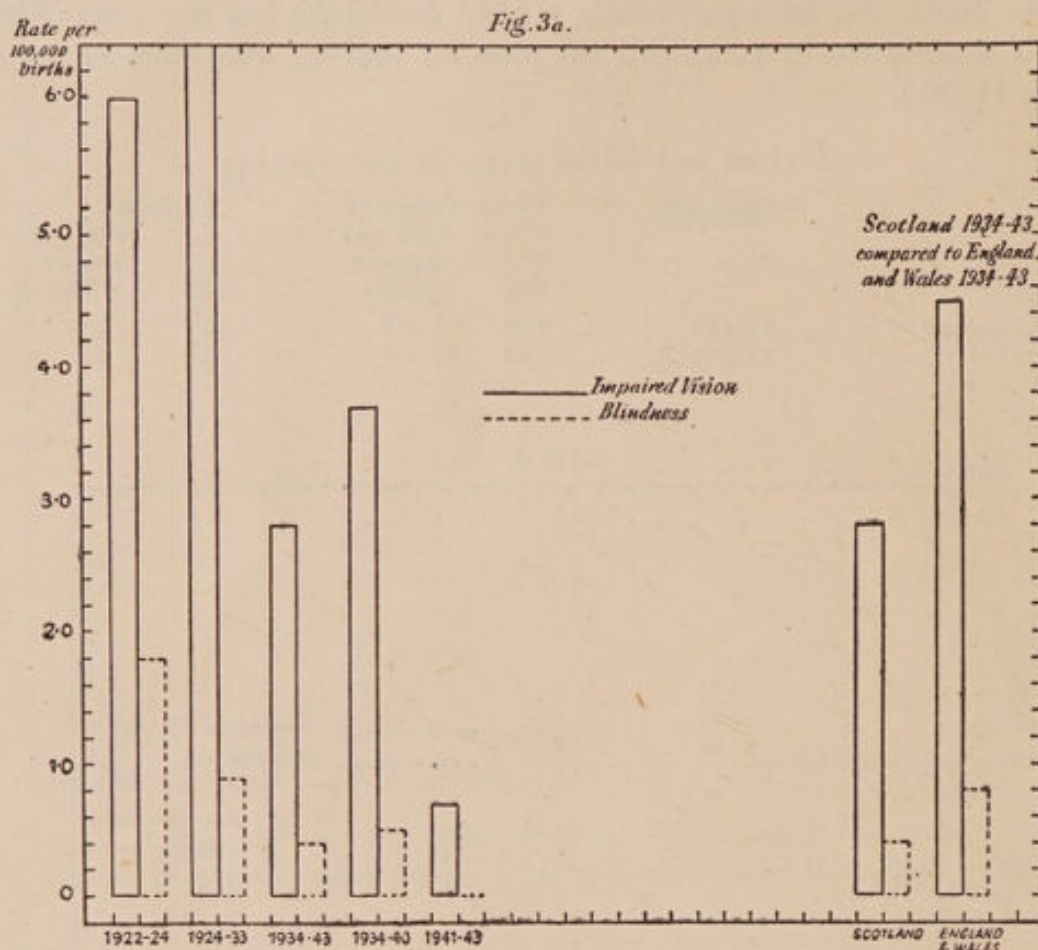
England and Wales, 1934-40 and 1941-43

	Notifications	Vision	Impaired Rate per 100,000 births	Blind	Blind Rate per 100,000 births
		No.		No.	
1934-40	29,410	190	4.5	44	1.0
1941-43	11,702	22	1.1	3	0.1



*Impaired Vision and Blindness from Ophthalmia Neonatorum.
Annual rates for England and Wales: 1934-43.*

For Scotland comparable data for the whole country are available from 1922 onwards (Table VI. See also Fig. 3a). Comparison of the two periods 1922-33 and 1934-43 shows the same tendency towards a decline.



*Impaired Vision and Blindness from Ophthalmia Neonatorum.
Scotland: 1922-43.*

Scotland, 1922-33 and 1934-43

	Notifications	Vision Appreciably Impaired		Blind	
		No.	Rate per 100,000 births	No.	Rate per 100,000 births
1922-33	16,531	70	5.9	12	1.0
1934-43	13,072	25	2.8	3	0.4

As in the case of England and Wales the decline in Scotland has been most marked during the last three years.

Scotland, 1934-40 and 1941-43

	Notifications	Vision Appreciably Impaired		Blind	
		No.	Rate per 100,000 births	No.	Rate per 100,000 births
1934-40	10,122	23	3.7	3	0.5
1941-43	2,950	2	0.7	—	—

This gratifying decline in the incidence of impaired vision and of blindness from ophthalmia neonatorum is reflected in the admissions to blind schools, where the decline in blindness from ophthalmia neonatorum has been marked for some years.

(1) *The Older Blind Statistics*

In 1881 Emrys-Jones found that 24 of the 72 candidates for admission to Henshaw's Blind Asylum had been blinded by ophthalmia neonatorum. An incidence of between 30% and 41% of inmates at blind schools and institutions was obtained in a collective survey conducted by the Ophthalmological Society in 1884. An incidence of the same order was reported by Snell in 1888 when he found that of 93 inmates in the Sheffield School for the Blind 37 (= 39.7%) were blind from this affection. Some twenty years later the position had not altered much. Of a total of 333 inmates who had passed through the school 136 (42.4%) had been blinded by ophthalmia neonatorum and "a careful consideration of the records for successive years does not even point to a diminution in more recent times" (Snell, 1907). At the Hull Blind Institute, Rockcliffe (1912) surveying 590 inmates over a period of 28 years found the position to be more hopeful; ophthalmia neonatorum was responsible for blindness in only 91 cases (15.4%) and was steadily declining. Of the 91 patients 53 were blind when first seen in 1884, 20 had been entered between 1884 and 1894, 16 in the next decade and only 2 between 1904 and December 1911. A decline in the incidence of ophthalmia neonatorum as a factor in blindness in children passed for blind schools in London in the two periods 1907-13 and 1914-20 is recorded by Harman (Ministry of Health, 1922). Of the total number admitted in the first period, 18.0%, and in the second period, 11.9%, had been blinded by ophthalmia neonatorum (134 out of 737 and 89 out of 755, respectively). Amongst 255 persons at the Swiss Cottage School for the Blind, of whom all but 16 were under the age of 25 years, Thompson in 1928 found 57 (22.3%) blinded by ophthalmia neonatorum.

(2) *Sunshine Homes for Blind Babies*

A clearer appreciation of the downward trend of ophthalmia neonatorum as a cause of blindness in children is obtained from the following data which refer to infants under the age of 5 years and contrast the situation in 1921 against that in 1943. In 1921 Harman reported on the causes of blindness in 63 babies at a "Sunshine Home" for blind infants conducted by the National Institute for the Blind. The home had then only recently been opened. To-day there are five such homes, which in January 1943 had 127 inmates. The contrast in the incidence of blindness from ophthalmia neonatorum in babies at Sunshine Homes in 1921 and 1943 is striking.

*Incidence of Blindness from Ophthalmia Neonatorum
at Sunshine Homes for Blind Babies*

Year	Number blind from Ophthalmia Neonatorum	Percentage of all blind babies
1921	31	49.2
1943	15	11.8

That the downwards trend has been steady for some years can be seen from Table VII, where the causes of blindness are given in the admissions year by year for the period 1920-43. (See also Fig. 4.) The following summary table brings out the salient features. (There is no reason to assume that the criteria for admission to the homes have changed during this period.)

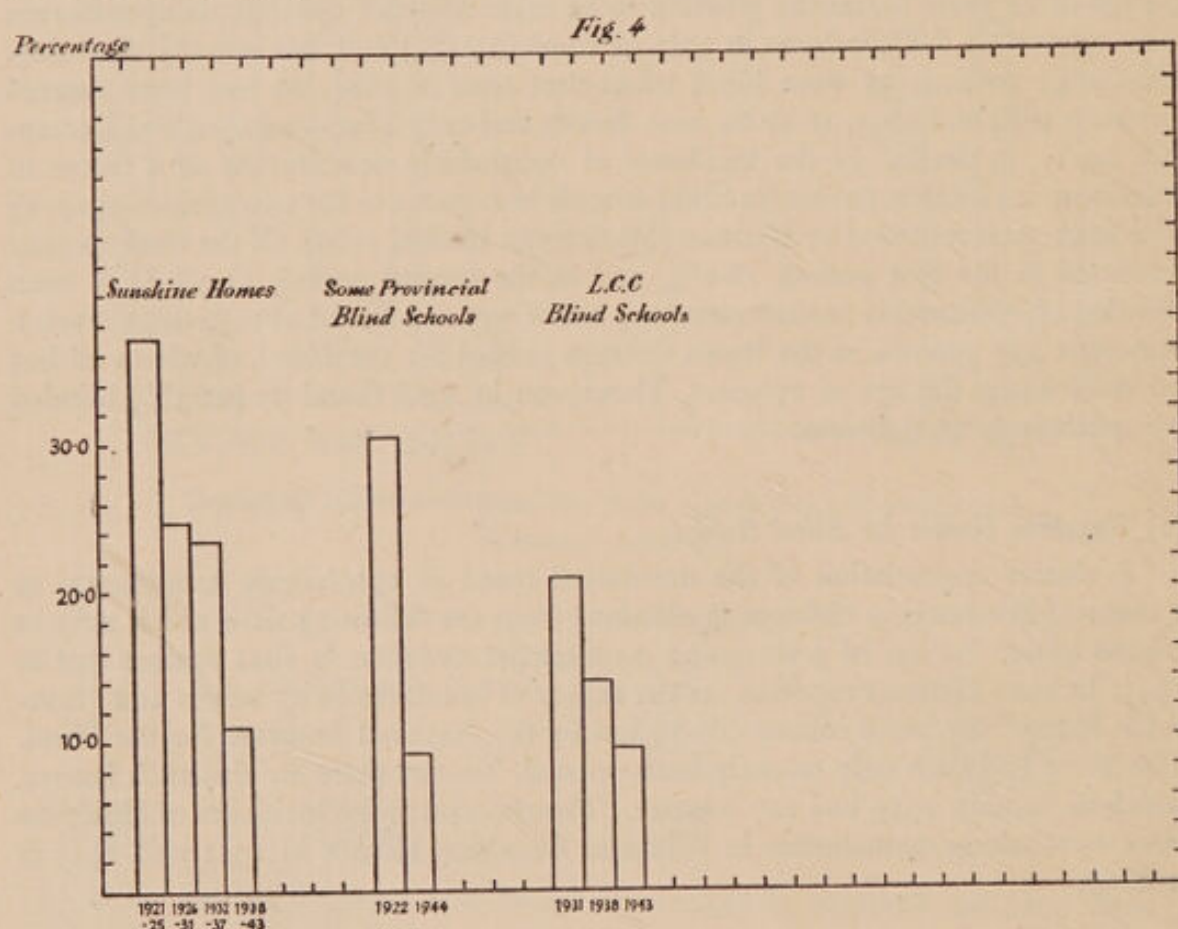
Sunshine Homes for Blind Babies

*Incidence of Ophthalmia Neonatorum in the Annual Admissions
for the periods 1920-25, 1926-31, 1932-37 and 1938-43*

Period	Number blinded from Ophthalmia Neonatorum	Percentage of all (fully certified) blind babies admitted
1920-25	43	37.1
1926-31	36	25.5
1932-37	33	23.9
1938-43	17	11.0

(3) *L.C.C. Blind Schools*

The diminished incidence of ophthalmia neonatorum as a cause of blindness can also be seen in its declining percentage of the total of children in L.C.C. blind schools (Fig. 4).



*Proportionate Incidence of Blindness from Ophthalmia Neonatorum.
In Blind Schools: 1921-44.*

*Incidence of Blindness from Ophthalmia Neonatorum in Pupils
at London County Council Schools for the Blind*

Year	Number blind from Ophthalmia Neonatorum	Percentage of all blind pupils
1913 ¹	[?]	24.2
1920 ¹	[?]	19.8
1931 ²	46	21.1
1938 ²	14	14.0
1943	5	9.8

¹ Harman, 1921.

² London County Council Annual Reports.

(4) *Provincial Blind Schools*

Comparative values for other blind schools are not readily available, but the following figures from Birmingham show the same trend.

Birmingham, Smethwick and West Bromwich Pupils admitted to the Kindergarten, Birmingham Royal Institution for the Blind, 1923-43 (exclusive of babies admitted to Sunshine Home)

	Number blind from Ophthalmia Neonatorum	Percentage of total
1923-32	14	26.9
1933-43	4	12.9

More substantial evidence on the decline of ophthalmia neonatorum as a cause of blindness in childhood is brought out by comparing the data on the causes of blindness at blind schools provided in 1922 by the Board of Education (Ministry of Health, 1922) and those shown in Table VIII. (See also Fig. 4.)

Incidence of Blindness from Ophthalmia Neonatorum at some Residential Blind Schools in the Provinces

Year	School	Number blind from Ophthalmia Neonatorum	Percentage of all blind pupils
1922	16 schools listed by Board of Education Return in Ministry of Health Report, 1922 (excluding Elm Court and Linden Lodge (L.C.C.) Schools)	282	30.4
1944	7 provincial schools shown in Table VIII.	48 (including 2 doubtful cases)	9.2

Summary Tables on the Decline of Ophthalmia Neonatorum as a cause of Blindness

The data on the immediate damage by ophthalmia neonatorum provided by the Ministry of Health and by the Department of Health for Scotland are entirely consistent with the findings recorded in the returns from Sunshine Homes for Blind Babies, the L.C.C. and the provincial schools for the blind. They all indicate a rapid decline in blindness from ophthalmia neonatorum during the past quarter of a century. Figs. 1-4 and the following summary bring out the salient features.

*Rates of Impaired Vision and of Blindness
per 100,000 Births*

		Impaired vision	Blindness
English County Boroughs	1924-33	18.7	2.2
	1934-43	4.5	0.9
Metropolitan Boroughs	1924-33	11.0	1.0
	1934-43	1.6	0.4
England and Wales	1934-40	4.5	1.0
	1941-43	1.1	0.1
Scotland	1922-33	5.9	1.0
	1934-40	3.7	0.5
	1941-43	0.7	0.5

*Proportionate Incidence of Blindness from Ophthalmia Neonatorum
amongst Children at Blind Schools*

		Percentage of Blindness due to Ophthalmia Neonatorum
N.I.B. Sunshine Homes	1920-25	37.1
	1926-31	25.5
	1932-37	23.9
	1938-43	11.0
L.C.C. Blind Schools	1913	24.2
	1920	19.8
	1931	21.1
	1938	14.0
	1943	9.8
Birmingham Pupils at Birmingham Royal Institution for the Blind	1923-32	26.9
	1933-43	12.9
Other Blind Schools	1922	30.4
	1944	9.2

3. REGIONAL VARIATIONS IN THE INCIDENCE OF IMPAIRED VISION AND OF BLINDNESS FROM OPHTHALMIA NEONATORUM

As the returns from the local authorities on the incidence of impaired vision and of blindness from ophthalmia neonatorum cover the whole of England and Wales since 1934, a comparative study for the different geographical areas is possible. The essential facts derived from Table IV, summarized in Table IV (6) and shown in Figs. 5, 6 and 7, are these:

*Incidence of Impaired Vision and of Blindness from Ophthalmia Neonatorum
in England and Wales, 1934-43*

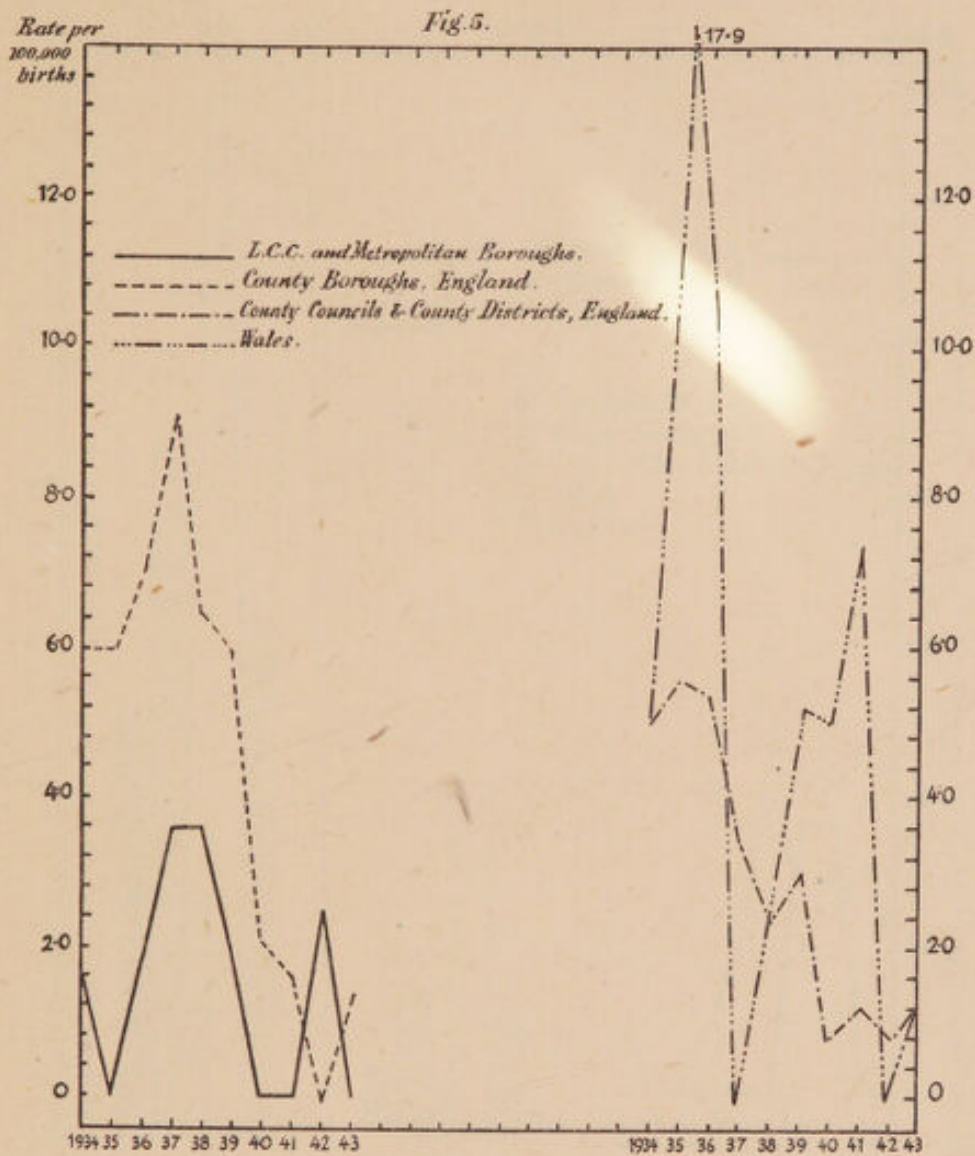
	Notifications	Impaired Vision		Blind	
		No.	Rate per 100,000 births	No.	Rate per 100,000 births
England:					
Administrative Counties	13,378	93	2.8	18	0.5
County Boroughs	21,901	90	4.5	18	0.9
L.C.C. and Metropolitan Boroughs	3,613	8	1.6	2	0.4
Wales (whole of)	2,220	21	5.3	9	2.3

London thus stands out favourably and the position of the County Councils is better than that of the County Boroughs, whilst the position of Wales is markedly unfavourable.

For the period 1924-33 comparative data are available for the Metropolitan Boroughs and the English and Welsh County Boroughs (Table III). Essentially the same comparative values obtain as for the fuller returns for England and Wales for 1934-43.

*Incidence of Impaired Vision and Blindness from Ophthalmia Neonatorum
in the L.C.C. Area and in County Boroughs, 1924-33*

	Notifications	Impaired Vision		Blind	
		No.	Rate per 100,000 births	No.	Rate per 100,000 births
Metropolitan Boroughs	6,898	76	11.0	7	1.0
English County Boroughs	27,566	394	18.7	46	2.2
Welsh County Boroughs	932	16	15.9	4	4.0



Impaired Vision from Ophthalmia Neonatorum.
Annual rates for England and Wales, 1934-43, by Administrative Areas.

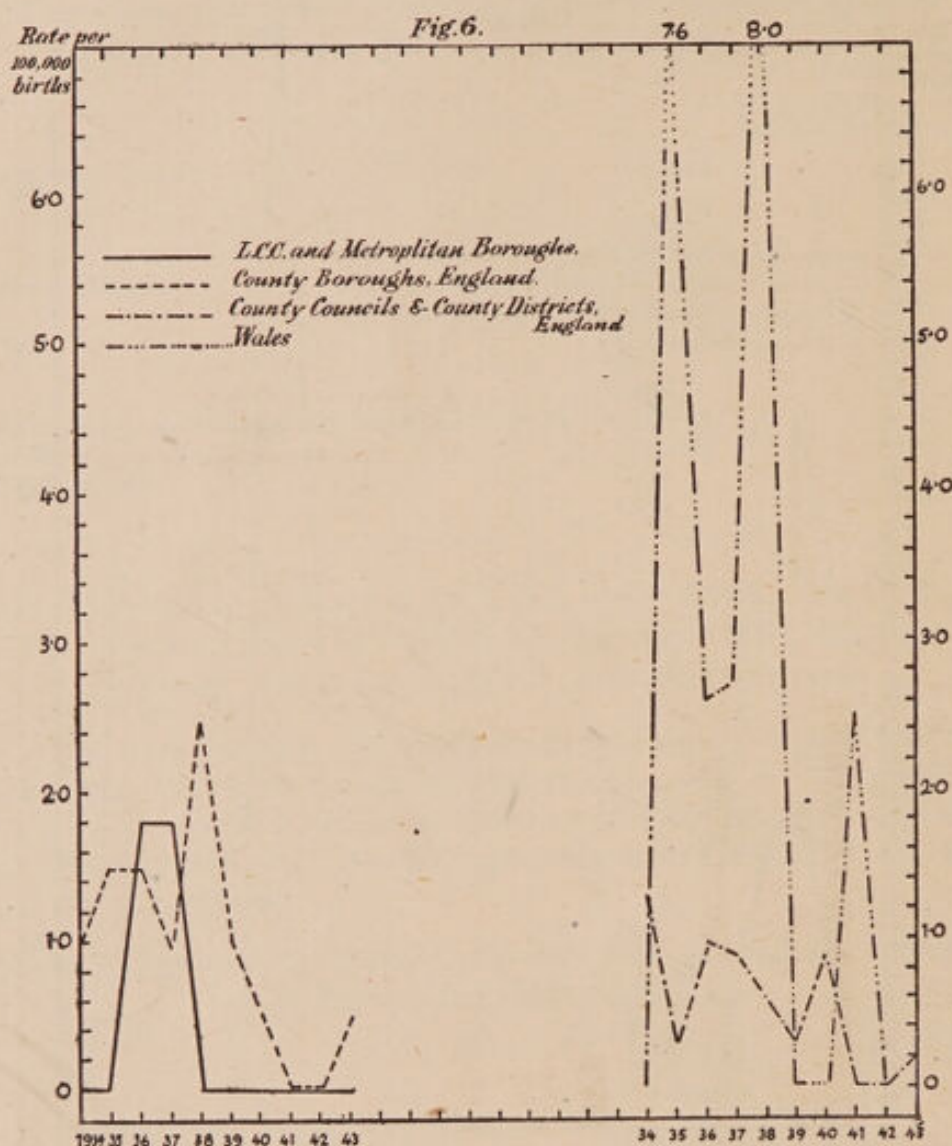
Comparison with the experiences of England and Wales as a whole [Table IV, (5)] with Scotland (Table VI) is of some interest.

*Incidence of Impaired Vision and Blindness in England and Wales
and in Scotland, 1934-43*

	Notifications	Impaired Vision		Blind	
		No.	Rate per 100,000 births	No.	Rate per 100,000 births
England and Wales	41,112	212	3.4	47	0.8
Scotland	13,072	25	2.8	3	0.4

It will be noted that the rates for Scotland as a whole are of the same order as those for London, and are distinctly better than those for England as a whole and very much better than those for Wales. (See also Fig. 3a.)

In contrast to London with its low notification rate is Birmingham with an exceptionally high rate, especially since 1934 (Table V). It is therefore of some



*Blindness from Ophthalmia Neonatorum.
Annual rates for England and Wales, 1934-43, by Administrative Areas.*

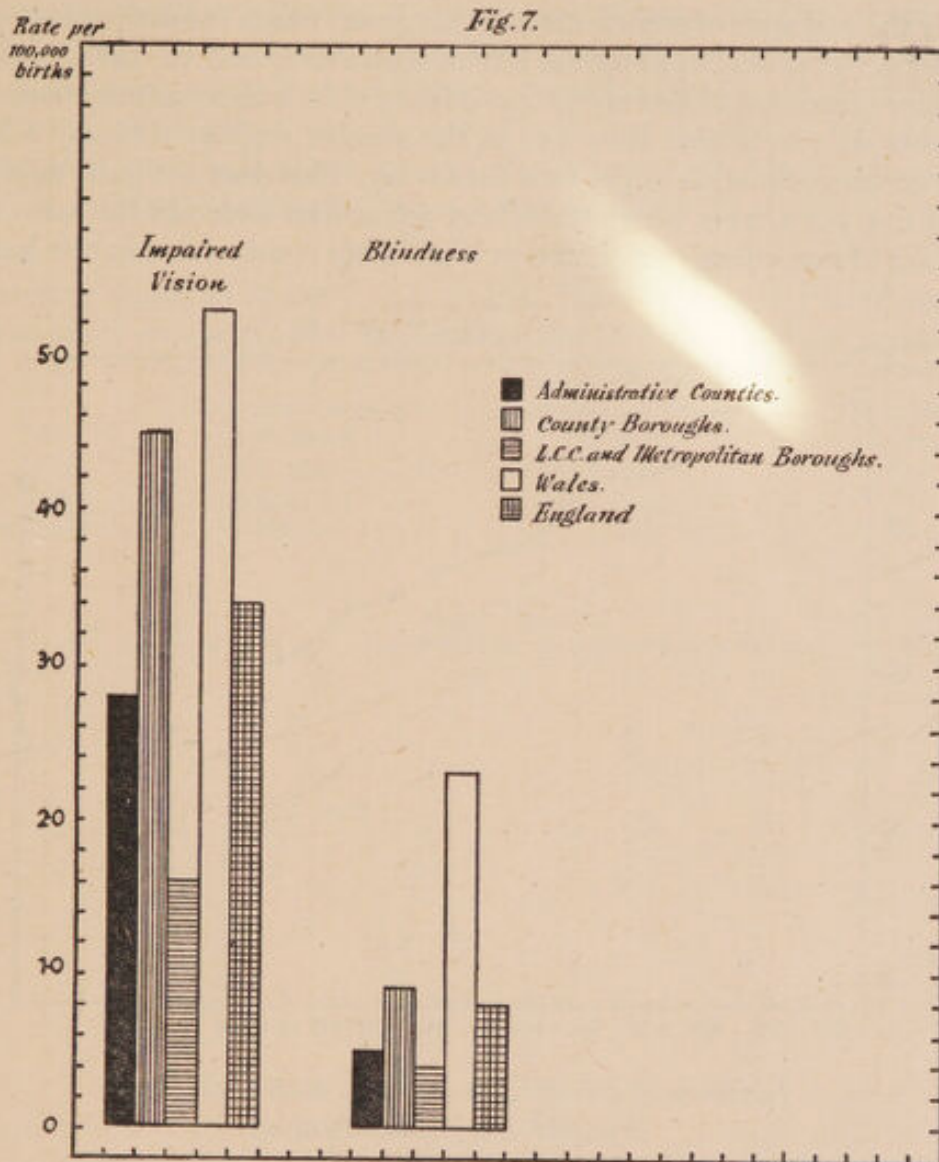
interest to compare the rates for impaired vision and blindness in these two areas (Tables III (1), IV (1), and V).

		<i>Impaired Vision</i>		<i>Blind</i>	
		No.	Rate per 100,000 births	No.	Rate per 100,000 births
Birmingham	1924-33	42	24.4	10	5.8
London	"	76	11.0	7	1.0
Birmingham	1934-43	3	1.7	0	0
London	"	8	1.6	2	0.4

4. THE DECLINE IN THE INCIDENCE OF BLINDNESS FROM OPHTHALMIA NEONATORUM IN RELATION TO BLINDNESS FROM OTHER CAUSES

(1) *Decline in Blindness in Childhood, 1923-43*

The returns obtained by the Ministry of Health of the number of blind persons since the introduction of the Blind Persons Act of 1920 show a steady increase in



*England and Wales by Administrative Areas.
Average rates for Impaired Vision and Blindness from Ophthalmia
Neonatorum: 1934-43.*

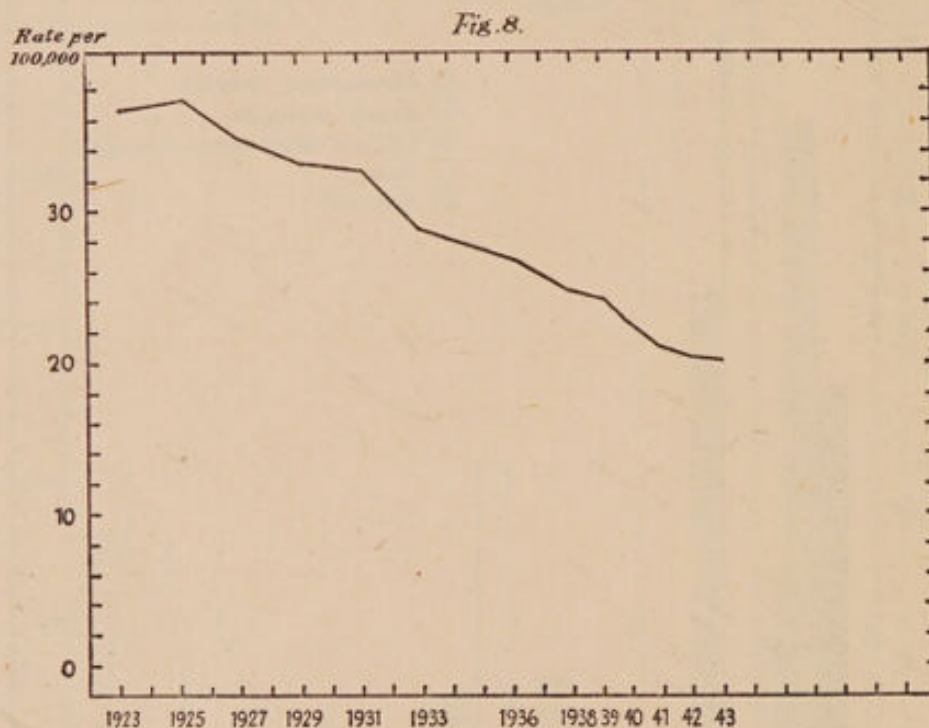
the number of registered blind in England and Wales. In 1919 it was 25,840; a year later 30,785. From 36,518 in 1923 it rose steadily to 63,408 in 1933 and to 76,507 in 1943. These figures represent, of course, an improvement in effective registration rather than an increase in the incidence of blindness itself. The increase in the number of registered blind has been largely confined to the advanced age groups, as can be seen from Table IX, from which the following summary table is extracted.

Age Distribution of Registered Blind in 1923 and 1943

Year	Age Groups						Unknown	Total
	0-5	5-16	16-21	21-50	50-70	over 70		
1923	231	2,723	1,567	10,955	12,397	8,026	619	36,518
1943	217	1,355	1,078	14,805	27,983	30,884	185	76,507

As far as the younger age groups are concerned the number of blind has shown a decline, not very marked for the age group 0-5 years but decidedly so for the age group 5-16 years, as can be seen from Table X. The figures for 5-16 probably show

more truly the real state of affairs, for it is this group which for many years now has been under active observation by the school authorities; and the initial number of blind children recorded in that age group is likely to be a close approximation to the total number who were then alive, just as the number registered to-day is likely to represent an approximation to the total for to-day. This does not quite apply to the age group 0-5 years, over which the school authorities have not the same control. The element of non-effective registration is probably considerable in this age group,



*Incidence of Blindness in Children aged 5-16 years.
England and Wales: 1923-43.*

though perhaps not so considerable as in the age groups over 16 years. Accepting, therefore, the figures for the age group 5-16 years as a true indication of the number of blind children in the country, an assessment of the significance of the smaller number registered in recent years becomes possible by computing the rate of blindness per 100,000. The following summary based on Table X (which is shown graphically in Fig. 8) brings out the steady downwards trend in the incidence of blindness.

*Incidence of Blindness in Children aged 5-16 years,
England and Wales, 1923-43*

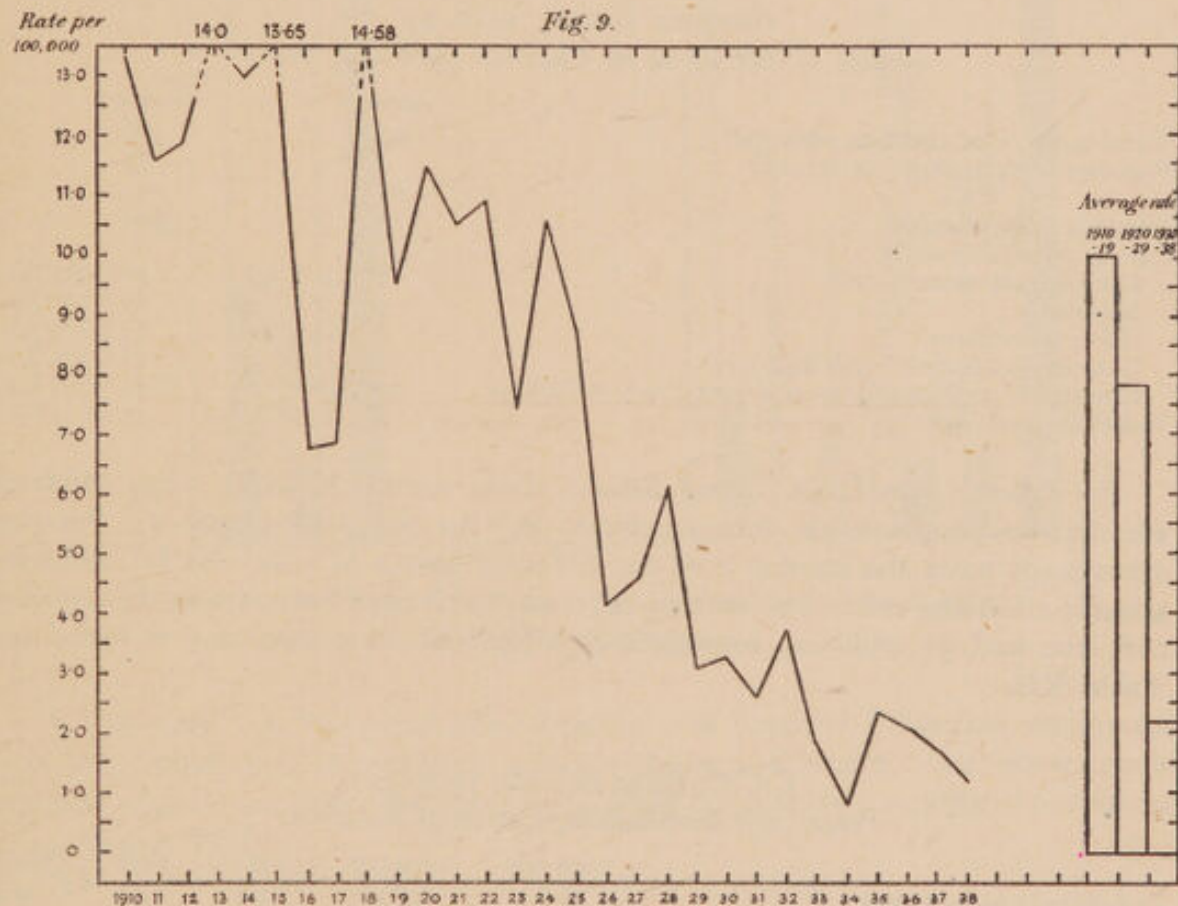
Year	Population aged 5-16 years	Number of registered blind in this age group	Rate per 100,000
1923	7,477,143	2,723	36.4
1933	7,246,446	2,089	28.8
1938	6,694,300	1,676	25.0
1942 ¹	—	1,369	20.5
1943 ¹	—	1,355	20.3

¹ Population figures for 1939 onwards are not available for publication. The rates of 20.5 blind in this age group in 1942 and 20.3 for 1943 are likely to be underestimates, as the population for 1938 was used in these computations.

An even more striking decline in the number of blind children of school age is recorded by the London County Council in their returns of admissions to blind schools. As these records refer to children between the ages of 3 to 16 years, they are not strictly comparable with the national returns, but the data of this large educational authority have an intrinsic interest. From 1910 there has been a continuous decline (Fig. 9), as the following figures based on Table XI show:

Incidence of Blindness in the L.C.C. School Population, aged 3 to 16 years, as determined by the Number of Pupils passed for Blind Schools, 1910-38

Year	Elementary school population	Number of children passed for blind schools	Rate per 100,000
1910-19	8,813,258	1,016	11.5
1920-29	7,912,164	624	7.9
1930-38	5,607,111	124	2.2



*Children requiring Education at a Blind School.
Annual Incidence in the L.C.C. School Population: 1910-38.*

It may therefore be accepted that the incidence of blindness in children has declined considerably during the past 20-30 years. The decline in blindness from ophthalmia neonatorum is undoubtedly a factor in this general reduction, and the question is whether and to what extent other operative causes of blindness have also declined.

(2) *Tentative Analysis of the Factors in this Decline*

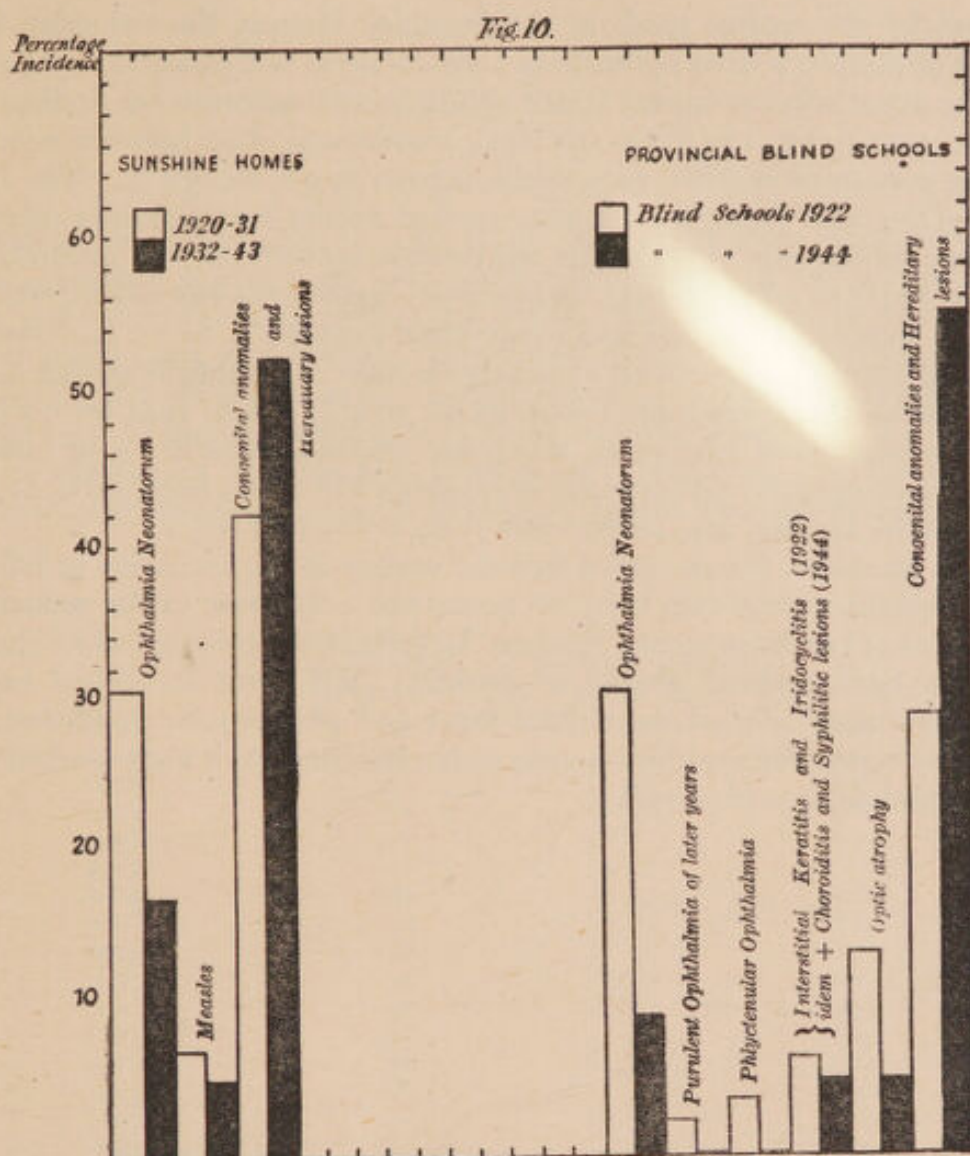
An analysis of the causes of blindness in the various age groups of the registered blind of the country is unfortunately not available, so that an assessment of any change in causation over a number of years cannot yet be made. The following incomplete information is of some significance (Fig. 10).

(a) *Causes of Blindness at Sunshine Homes, 1920-43.* The following summary table is based on Table VII and shows the causes of blindness during the twelve years 1920-31 contrasted with the subsequent twelve years 1932-43.

<i>Sunshine Homes, 1920-43.</i> <i>Causes of Blindness in Children aged 0-5 years</i>		
	1920-31	1932-43
Total number of children admitted	284	316
Number with incomplete records	27	24
Number fully certified	257	292
Causes of blindness:		
Ophthalmia neonatorum	79 (30.7%)	50 (17.1%)
Measles	18 (7.0%)	15 (5.2%)
Other infections	36 (14.0%)	29 (9.9%)
Trauma (including birth injuries)	4 (1.6%)	12 (4.1%)
Congenital malformations and hereditary affections	108 (42.0%)	153 (52.4%)
Indefinite aetiology	12 (4.7%)	33 (11.3%)

(b) *Returns from L.C.C. Blind Schools.* Data are available for 1,855 children admitted to blind schools during 1905-21 and for 369 during 1922-27. For the subsequent years the incidence of the different causes of blindness for children actually attending at blind schools in 1931, 1938 and 1943 has to be used (covering 218, 100 and 51 children, respectively). The following summary is based on Table XII.

<i>L.C.C. Blind Schools, 1905-43</i> <i>Percentage Distribution, Causes of Blindness</i>					
	1905-21	1922-27	1931	1938	1943
Ophthalmia neonatorum	19.8	11.3	21.1	14.0	9.8
Purulent conjunctivitis of later years	4.8	—	—	—	—
Phlyctenular ophthalmia and superficial keratitis	3.5	8.1	5.5	3.0	—
Interstitial keratitis and iridocyclitis	22.3	16.0	9.2	1.0	2.0
Optic atrophy	18.8	14.1	19.8	16.0	15.7
Injury	1.6	1.5	1.4	4.0	5.8
Congenital and hereditary lesions, including myopia	29.1	49.0	42.2	56.0	58.8
Other defects	—	—	0.9	6.0	7.9



Changes in the Proportionate Incidence of some Causes of Blindness in Childhood: 1920-44.

(c) *Returns from Provincial Blind Schools.* The Board of Education returns for 927 blind children at 16 provincial schools (Ministry of Health, 1922) allow a comparison with an analysis obtained for 524 blind children at 7 provincial schools in 1944 (Table VIII).

The following main groups lend themselves to comparison.

	Board of Education, 1922 927 cases	Present Series 524 cases (1944)
Ophthalmia neonatorum	30.4%	9.2%
Purulent ophthalmia of later years	2.5%	—
Phlyctenular ophthalmia	3.7%	0.2%
Interstitial keratitis	4.1%	0.9%
Iridocyclitis	2.5%	0.8%
Choroiditis	[?]	0.8%
Syphilitic lesions	[?]	2.5%
Optic atrophy	12.9%	5.3%
Congenital anomalies:		
Including myopia	37.2%	67.6%
Excluding myopia	30.8%	55.0%

Judged by the returns made at the Sunshine Homes, the reduction in the incidence of blindness from ophthalmia neonatorum is not paralleled by a similar decline for other infective causes; whilst ophthalmia neonatorum has declined from 30.7% (in 1920-31) to 17.1% (in 1932-43), measles and other infective conditions (including congenital syphilis) have declined from 21.0% to 15.1%. The L.C.C. figures, on the other hand, show a more marked decline for conditions other than ophthalmia neonatorum; thus whilst ophthalmia neonatorum has declined from 21.1% (in 1931) to 9.8% in 1943, phlyctenular ophthalmia has fallen from 5.5% to nil, and interstitial keratitis and iridocyclitis from 9.2% to 2.0% during that period. The returns for provincial blind schools show an essentially similar decline: in 1922 ophthalmia neonatorum accounted for 30.4%, and in 1944 for 9.2%, but purulent ophthalmia of later years, which was responsible for 2.5% of cases, had disappeared completely, phlyctenular ophthalmia had fallen from 3.7% to 0.2%, and interstitial keratitis and iridocyclitis from 6.6% to 1.8%.

It may, therefore, be concluded that the decline in the incidence of blindness from ophthalmia neonatorum is by no means the only cause in the reduction of the incidence of blindness among children. In general, infective causes of blindness in children have declined almost as markedly, and some individual causes—especially purulent ophthalmia of later years and phlyctenular ophthalmia, and possibly also congenital syphilis—appear to have declined to a more marked extent than has ophthalmia neonatorum.

CLINICAL

I. AETIOLOGY

WHATEVER may have been the case in the past, ophthalmia neonatorum is not entirely or mainly synonymous with gonococcal infection. This is shown by the relative low incidence of gonococcal ophthalmia recorded in recent years for large series of ophthalmia neonatorum. Admittedly, there is the possibility that the definition of ophthalmia neonatorum as used in practice to-day may be less stringent than that used by earlier observers, but the cumulative evidence as to a lower proportionate frequency of gonococcal ophthalmia is imposing. Indicative of many other findings of the same order, the following figures from Browning (1936) may be quoted.

Year	Total number of ophthalmia neonatorum cases	Number of gono- coccal cases	Percentage
1914	37	20	54.0
1915	50	22	44.0
1916	36	16	44.4
1917	12	10	83.3
1918	10	1	10.0
1919	?	?	?
1920	56	17	29.8
1921	78	13	16.6
1922	76	13	16.2
1923	39	5	12.2
1924	40	7	17.5
1925	44	6	13.6
1926	70	7	10.0
1927	66	1	1.5
1928	45	10	22.2
1929	57	5	8.7
1930	61	5	8.2
1931	53	2	3.7
1932	69	5	7.2
1933	47	3	6.4
1934	47	4	8.5
1935	37	0	0
	1030	172	16.7

Browning's series has some element of selection. Not all the cases of ophthalmia neonatorum seen at the Royal London Ophthalmic Hospital were sent for bacteriological examination, and it is possible that the more severe cases were admitted to St. Margaret's Hospital. The records of this latter hospital showed an incidence of gonococcal ophthalmia of about 60%, according to Mayou writing in 1931. St. Margaret's Hospital has been housed at White Oak Hospital (as a war measure) since September 1939, and from that date till December 1944 the case incidence of gonococcal ophthalmia has been distinctly lower than that recorded by Mayou. The distribution of organisms in the 737 cases admitted in that period is as follows:

*Incidence of Causative Organisms in Consecutive
Cases of Ophthalmia Neonatorum*

(September 1939 to May 8th, 1945)

No organism in smear or culture	126
Gonococcus	180
Staphylococcus:	
Staphylococcus (not specified)	105
<i>Staphylococcus aureus</i>	63
<i>Staphylococcus albus</i>	46
<i>Staphylococcus aureus</i> and diphtheroids	7
<i>Staphylococcus albus</i> and diphtheroids	4
Staphylococcus and diphtheroids	46
Other coccal organisms:	
Gram positive (not specified)	8
Meningococcus	6
Pneumococcus	9
<i>Streptococcus haemolyticus</i>	7
Non-haemolytic streptococcus	1
<i>Streptococcus viridans</i>	1
<i>Micrococcus tetragenus</i>	3
<i>Micrococcus catarrhalis</i>	3
Gram positive: unidentified	2
Gram negative: unidentified	1
Bacilli:	
Diphtheroids	60
Koch-Weeks	11
Coliform	9
Xerosis	3
Morax-Axenfeld	8
Friedlander	7
Hoffmann	2
Gram positive } (not specified)	{ 5
Gram negative }	{ 13
Pyocyaneus [? contamination]	1
	<hr/>
	737

The large proportion of cases in which no causal organism could be found—shown in this table and in most published series—at one time constituted a considerable puzzle, but it is now clear that the bulk of such cases represent not faulty bacteriological findings but examples of ophthalmia neonatorum due to a virus infection. It was recognized as early as 1909 that both in bacteriologically negative ophthalmia neonatorum and in cases of gonococcal ophthalmia intracellular bodies in the cytoplasm of the conjunctival epithelium could be found. These inclusion bodies, as they came to be known, raised the question as to the relationship between ophthalmia neonatorum and trachoma, in which affection similar inclusion bodies had been observed two years earlier by Halberstaedter and Prowazek. It is only of late years that any substantial addition has been made to knowledge gained in the brilliant and intensive pioneer work carried out around 1909–11. The incidence of inclusion blenorrhoea is variously estimated at between 8·8% and 34% of all ophthalmia neonatorum. That inclusion bodies may be found together with various organisms is clearly established, though there is no agreement upon the frequency of such association. Uncomplicated inclusion blenorrhoea is generally regarded as having a longer incubation period than ophthalmia neonatorum due to microbial organisms. In no instance did it occur before the 5th day, and only occasionally was it delayed beyond the 10th day in the 57 cases studied by Thygeson and his associates. It is generally agreed that there are no distinguishing features in the clinical appearance of inclusion blenorrhoea in comparison to organismal ophthalmia neonatorum. As in the case of trachoma virus, attempts to cultivate it have

failed, but in contrast to trachoma there is general agreement that the sulphonamides are effective in treatment. The genital origin of inclusion blenorrhoea was demonstrated in the early investigations, and has been confirmed by recent observers, who have found presumptive evidence of the presence of the virus in the cervix of women and the urethra of men. At White Oak Hospital inclusion bodies have been found 39 times in the last 415 cases of ophthalmia neonatorum. In no case was the gonococcus found and in only 6 cases were other associated organisms observed, the organisms being Koch-Weeks bacilli and haemolytic streptococci in one case each, and *Staphylococcus aureus* in 4 cases. In 32 mothers scrapings of the cervix were taken and on 9 occasions inclusion bodies could be demonstrated. That no inclusion bodies were seen in the remaining 23 mothers does not exclude the possibility that the virus might have been demonstrated by other methods than histological examination (such as inoculation into the conjunctiva of baboons). That these negative findings are not too significant is apparent from the fact that in a number of mothers, whose babies did not show inclusion bodies, such bodies were none the less found in scrapings from the cervix. The incubation period in these 39 cases was less than 5 days in 5 cases, as can be seen from the following table:

Onset on	Inclusion bodies	Inclusion bodies and organisms
2nd day	1	—
3rd day	3	—
4th day	1	1 (Koch-Weeks)
5th day	3	—
6th day	4	—
7th day	4	1 (<i>Staphylococcus aureus</i>)
8th day	5	1 (Haemolytic streptococci)
9th day	4	2 (<i>Staphylococcus aureus</i>)
10th day	3	—
11th day	2	—
12th day	4	—
13th day	1	—
15th day	1	—
18th day	1	1 (<i>Staphylococcus aureus</i>)
20th day	1	—
23rd day	1	—
	39	6

It is worth noting that two of the three cases in which the ophthalmia developed on the third day showed inclusion bodies in both the baby and mother, and were in addition bacteriologically negative.

A fuller account of inclusion blenorrhoea is given elsewhere (Sorsby, Hoffa and Young, 1944).

Judging by the material at White Oak Hospital, the causative factors in ophthalmia neonatorum are

Gonococcus	in about 25%
Staphylococcus	" " 35%
Other coccal organisms	" " 5%
Various bacilli	" " 20%
Virus	" " 10%
Undetermined	" " 5%

2. PROPHYLAXIS

First, to remove, if possible, the disease in the mother during pregnancy; secondly, if that cannot be accomplished, to remove artificially as much of the discharge as possible from the vagina at the time of delivery; and third, to pay, at all events, particular attention to the eyes of the child by washing them immediately after delivery with a liquid calculated to remove the offending matter or to prevent its noxious action.

BENJAMIN GIBSON,
Edin. Med. and Surg. Jl., 1807, p. 160.

(1) *Obstetric Asepsis*

THE programme outlined by Gibson at the beginning of the last century could not be translated into reality until, in the first place, vaginal infection as the exclusive and not merely a contributory cause or one of many possible causes of ophthalmia neonatorum had become accepted teaching—and this did not happen till well beyond the middle of the century—and, secondly, until adequate means to combat that infection in the mother and her baby had become available. That Credé, a child of the Listerian age of antisepsis, should have sought to control the infection by antiseptics is natural enough, and it is only in more recent years that the first desideratum laid down by Gibson has gained full recognition.

Credé's essays in antisepsis concentrated at first on cleansing the vaginae of pregnant women and of women actually in labour. The results were not altogether satisfactory, and whilst continuing that practice he sought to reinforce the disinfection by applying antiseptics to the eyes of the new-born, as others before him had done. Silver nitrate, which was freely used—and indeed abused—throughout the nineteenth century in inflammation of the eyes proved to be a satisfactory local disinfectant and was presented by him as the only method of prophylaxis (apart from swabbing of the lids).

The immense reduction in the incidence of ophthalmia neonatorum that followed the widespread attention to Credé's teaching has tended to focus attention away from the considerable residue of ophthalmia neonatorum that has persisted. This residue was regarded as the exception that proved the rule of the efficacy of local antisepsis and the search for a more potent—and more fool-proof and less irritating—antiseptic than silver nitrate overshadowed the need for obstetric asepsis. It is likely that the incidence of ophthalmia neonatorum in hospital practice is to-day no lower than it was some 40 to 50 years ago. The incidence of ophthalmia neonatorum in 35,815 births at 5 London lying-in hospitals and at the Rotunda Hospital, Dublin, was given by Stephenson (1907) as 0.22% for various years between 1889 and 1906, whilst to-day the incidence of gonococcal ophthalmia alone is of the order of 0.08% as determined by returns on 221,131 births in 45 lying-in centres (recorded on p. 33).

The need of ante-natal care is of course an obstetric axiom for reasons other than the prevention of ophthalmia neonatorum. How significant ante-natal care is in the prevention of ophthalmia neonatorum is seen from the following experiences:

(1) Lehrfeld (1935). In 27,873 births at Philadelphia hospitals there were 632 cases (2.2%) of ophthalmia neonatorum, of which approximately 30% were gonococcal. At hospitals where careful treatment of gonorrhoea of the expectant mother was carried out, the incidence of ophthalmia neonatorum was one-fifth that obtain-

ing at hospitals where no such preliminary treatment was undertaken. At two hospitals where routine procedures were carried out to discover the presence of gonorrhoea during pregnancy the rate of ophthalmia neonatorum was 7 and 10 per 1,000 babies born respectively, against rates of 36 and 61 at two other institutions where this routine was not adopted.

(2) Castallo and Feo (1940). At Jefferson Medical College Hospital, Philadelphia, there were 7,116 births during 1927-38. Gonorrhoea in the mother was diagnosed in 200 cases before labour, but only in 170 was adequate local treatment carried out. None of the babies of these 170 women developed gonococcal ophthalmia, whilst there were 4 cases in the babies of the untreated mothers. There were in addition 18 babies with gonococcal ophthalmia neonatorum born from mothers in whom gonorrhoea had not been diagnosed before labour. (In addition to these 22 cases of gonococcal ophthalmia neonatorum amongst the 7,116 babies there were 24 cases of non-gonococcal ophthalmia neonatorum.)

(3) Fairfield (1943). In 1937 there were 19,222 live births and 722 still births in the maternity units of the London County Council hospitals; in 17 cases gonococcal ophthalmia neonatorum was observed. Of these 17 cases, 9 occurred amongst the 17,936 "booked" births (live and still), i.e. the mothers had previously received ante-natal care. The remaining 8 cases occurred amongst 1,625 emergency admissions (in these cases the mothers had had no ante-natal care or had received it from other than the Council's services). The rate of incidence of gonococcal ophthalmia in the two series was therefore 0.5 per 1,000 and 5 per 1,000 respectively. None of the 17 mothers concerned was known to have a history of gonorrhoea; some had vaginal discharge, but a bacteriological examination proved negative to gonococci, though 13 (and possibly 15) were found to be gonococcus positive after delivery.

(4) Sheffield Street Hospital. This hospital is the London County Council's obstetric unit for mothers with known venereal disease. During 1940-42 there were 210 births to mothers all suffering from gonorrhoea, and 3 cases of gonococcal ophthalmia neonatorum were observed. These three babies were, however, born before the mothers had been admitted to the hospital. Four cases of non-gonococcal ophthalmia (2 staphylococcal, 2 in which no organism could be recovered) were observed in this series. Silver nitrate (1%) prophylaxis is used as a routine measure at this hospital in addition to ante-natal treatment.

These findings emphasize the importance of ante-natal treatment in the prevention of gonococcal ophthalmia neonatorum and suggest that silver nitrate does not prevent the onset of non-gonococcal ophthalmia neonatorum. The stress laid on the ante-partum treatment of maternal gonorrhoea is both valid and necessary, but it must also be remembered that organisms other than gonococcus and at least one virus may produce ophthalmia neonatorum. The treatment of maternal leucorrhoea of non-gonococcal origin is therefore as important as that of maternal gonorrhoea, especially as there is some evidence that silver nitrate prophylaxis is more effective against gonococci in the conjunctiva of the new-born than against staphylococci and the other exciting agents of ophthalmia neonatorum.

The prevention of ophthalmia neonatorum lies in the first place in the treatment of the expectant mother, and this would as yet seem to be an aspiration rather than the reality. How much of an aspiration and how little of a reality is only too clearly shown by the fact that the majority of births in this country do not have the benefit of medical attendance, while ante-natal attention is still the exception rather than the rule.

(2) *Conjunctival Disinfectants*

The efficacy of conjunctival disinfectants is suggested by a collective investigation of Kay Sharp and others undertaken for the Northern Counties Association for the Blind (1935). In 37 areas having 1,955,349 births during the years 1924-33, various silver preparations were used prophylactically as a routine by the midwives employed by the local authority; in 7 areas they were not used. The rate of ophthalmia neonatorum notified per 1,000 births in these two sets of areas were:

	Areas using prophylactic drops	Areas not using such drops
1924	9.9	17.5
1925	8.9	18.2
1926	8.5	17.8
1927	8.6	19.3
1928	8.2	16.4
1929	7.3	18.0
1930	7.2	18.3
1931	6.1	20.0
1932	6.3	18.7
1933	6.0	18.2

But as notifiable ophthalmia neonatorum is not a clearly defined entity these data have to be interpreted with some reserve, especially as there is no information on other possible contributory factors, nor on the relative distribution of gonococcal ophthalmia neonatorum in the two series.

(a) *Silver Nitrate*. Though used for over fifty years and well established as an antiseptic agent in the conjunctival sac (Ridley, 1931), knowledge of the ocular pharmacology of silver nitrate still leaves much to be desired. Generally regarded as a specific against the gonococcus, some have doubted this (Gottlieb and Freedman, 1934), and the view has even been advanced that silver nitrate stimulates rather than inhibits the gonococcus (Cecchetto, 1937).

In many continental countries silver nitrate prophylaxis is compulsory at all births. Unfortunately, statistical data on the incidence of ophthalmia neonatorum and its sequelae are scanty. Some possibility of assessing the value of silver nitrate as a prophylactic is offered by the practice obtaining in Scotland. The Scottish Central Midwives Board, unlike the C.M.B. for England and Wales, requires midwives to use 1% silver nitrate as a routine. The practice is not legally compulsory at all births but is apparently very widely carried out, and the following comparative table on the incidence of visual defect and of blindness arising from ophthalmia neonatorum (based on Tables IV (5) and VI) is of some interest.

	Visual Impairment Rates per 100,000 births	Blindness
1934-43		
England and Wales	3.4	0.8
Scotland	2.8	0.4

But to ascribe the better results obtained in Scotland solely to silver nitrate prophylaxis is to ignore the possibility of other factors being operative.

Indirect proof of the efficacy of silver nitrate as a conjunctival disinfectant is contained in the following findings reported by Flack (1942). At the Sloane Hospital for Women (Columbia Presbyterian Medical Centre) there were 11 cases of gonococcal ophthalmia in 16,922 births during 1929-36 where the procedure consisted of the instillation of 1% silver nitrate followed by irrigation with normal saline. In contrast, only one case occurred in 10,248 births during 1936-40 when washing out the silver nitrate by irrigation was given up.

Some of the disadvantages of silver nitrate have been recognized from the time it was first employed empirically in the eighties of the last century. Apart from the occasional tragedies that have occurred from this colourless solution being confused with other reagents, it is the general experience that the drug is apt to be irritating and may produce a catarrhal conjunctivitis. Evaporation and decomposition by light are important causes of fairly rapid changes in the concentration and acidity of the solution. The recognition of these factors, and of the danger of confusion with other solutions, has led to the marketing of fairly stable preparations in glass capsules (Hellendall, 1930), and beeswax capsules, preferably lined with paraffin (Bunney, 1935). Concentration and pH level remain constant for much longer periods in such containers.

(b) *The Substitutes of Silver Nitrate.* To test the relative value of silver nitrate and other drugs as prophylactics a questionnaire was addressed to 67 of the more important obstetric centres in the country. Information was sought on the following points for the five-year period 1938-42:

- (1) Number of live births.
- (2) Total number of cases of ophthalmia neonatorum.
- (3) Number of cases specifically gonococcal in character.
- (4) Whether silver nitrate was used as a routine.
- (5) If not, what other routine prophylactic, if any, was used.

At only one small centre were prophylactic drops not used, and no information was available on the number of cases of gonococcal ophthalmia. As some of the replies were incomplete, the conditions obtaining at 45 centres are summarized in the following table.

*Incidence of Gonococcal Ophthalmia Neonatorum in Births
at 45 Obstetric Centres during 1938-42*

	Number of births	Number of cases	Rate per 1,000 births
Routine use of silver nitrate prophylactic (26 centres)	128,266	122	1.0
Preparations other than silver nitrate (20 centres)	92,865	62	0.7
	221,131	184	0.8
Centres not using silver nitrate as a routine, but using			
Argyrol (5-20%) (12 centres)	60,603	43	0.7
Protargol (4 centres)	14,392	3	0.2
Flavine in castor oil (1 : 1,500) (1 centre)	6,287	8	1.2
Acriflavine in castor oil (1 : 10,000) (1 centre)	4,333	—	—
¹ Mercury perchloride (1 : 4,000) (1 centre)	2,970	5	1.7
Mercury perchloride (1 : 10,000) (1 centre)	4,280	3	0.7
	92,865		

¹ Discontinued. Changed over to silver nitrate. Hence this centre appears twice in this list.

In assessing the efficacy of a prophylactic agent, gonococcal ophthalmia neonatorum rather than ophthalmia neonatorum as a whole is used in this table, as the incidence of gonococcal cases was fairly consistent throughout the returns, but the incidence of ophthalmia neonatorum as a whole showed wide fluctuations (see page 11).

If these figures are to be taken at their face value, there is no tangible difference in efficacy between silver nitrate and at least one of the organic silver substitutes—

argyrol. This conclusion could only be valid if the ante-natal care and other relevant factors operated equally in the two groups of obstetric units. In the absence of such definite knowledge the conclusion can be only tentative.

Inconclusive as much of the evidence on the value of disinfection of the conjunctiva is, it appears, however, that

- (1) The use of conjunctival antiseptics is beneficial.
- (2) The disadvantages in the use of silver nitrate are largely accidental and not inherent in the reagent. If supplied in suitable containers (paraffin-lined beeswax capsules) it is relatively stable and foolproof.
- (3) There is no evidence that silver nitrate is superior to some of the organic silver preparations, such as argyrol, whilst it undoubtedly calls for more care in its use if undesirable and even disastrous sequelae are to be avoided.

3. SULPHONAMIDE THERAPY

THE promise of sulphanilamide in the treatment of ophthalmia neonatorum gained early and wide recognition. Newman (1937), Fernandez and Fernandez (1938), Perry (1938), among American observers, and Magitot *et al.* (1938), Dollfus *et al.* (1938), Pagès and Duguët (1938) among French observers, reported good results with sulphanilamide, whilst a sulphanilamide derivative, uleron, was employed by Best (1938) and Jess (1938) in Germany and by Szinegh (1939) in Hungary. With the introduction of sulphapyridine, attention was concentrated on this drug because of its selective action on the gonococcus, and numerous observers have reported highly gratifying results. These refer to relatively small groups of cases. In this country the value of sulphapyridine has been stressed by Michie and Webster (1938), Moffatt (1940), Clancy (1941), and Somerville-Large (1941). The medical officer of health for Glasgow in his annual reports for 1938 and 1939 noted that the result of the treatment of gonococcal ophthalmia neonatorum with sulphapyridine "was dramatic. Within twenty-four hours the clinical condition of the eyes had cleared up. . . . Most cases needed only two or three days' treatment of the drug, and the average stay in hospital for a gonococcal case has now been reduced from about eight weeks to ten days."

No intensive studies of the comparative value of the various sulphonamides are available. Mullen (1942) has reported on the use of sulphathiazole in 5 cases of gonococcal ophthalmia. A series of 40 cases—20 treated with sulphanilamide, 10 with sulphapyridine and 10 with sulphathiazole—has been recorded by Wong (1942). The study by Lewis (1941) is concerned mainly with sulphanilamide used in 90 cases and found inferior to sulphapyridine used in 22 cases and to sulphathiazole used in 8 cases of gonococcal ophthalmia. Sweet (1942) gives a comparative study of 32 cases treated with sulphanilamide, 50 with sulphapyridine, 8 with sulphathiazole and 16 with sulphadiazine. Smaller comparative studies are also given by Blumberg and Gleich (1943).

Most observers speak only of gonococcal ophthalmia, but sulphapyridine is equally effective in all forms of ophthalmia neonatorum (Somerville-Large, 1941; Sorsby, Hoffa and Smellie, 1942; Sorsby and Hoffa, 1944; Sorsby and Hoffa, 1945).

Several observers have reported on the use of sulphanilamide or sulphapyridine applied locally (Rein and Tibbetts, 1939; Bruens, 1940; Pillat, 1940; Guyton, 1941; Lewis, 1941; Panneton, 1941). It would appear that oral administration gives distinctly better results.

Experience at White Oak Hospital

St. Margaret's (L.C.C.) Hospital for ophthalmia neonatorum was transferred to White Oak Hospital on the outbreak of war. From September 1939 to May 8th, 1945, 737 cases were treated; 46 by the classical local methods during 1939; 606 by general sulphonamide therapy; and 85 by local application of penicillin.

The 606 cases treated by sulphonamides fall into two distinct groups: 273 cases observed during 1940-41 and 333 cases in 1942-44. The first series were treated by sulphapyridine, but there was considerable experimentation as regards optimum dosage; it was only towards the end of the series that a standard dosage had been established. The second series all obtained a uniform dosage and consisted of 156 cases treated with sulphapyridine, 84 with sulphamezathine, 47 with sulphathiazole, 32 with sulphadiazine, and 14 with sulphanilamide.

(1) Experimental Series of 273 Cases Treated with Sulphapyridine

This series may be dismissed briefly, and the subjoined table shows the obvious superiority of general sulphapyridine treatment over treatment by the local classical methods.

<i>Clinical Cure</i>	<i>Local Treatment (Classical Methods)</i>			<i>General Sulphapyridine Treatment</i>		
	Gonococci- positive	Gonococci- negative	Total	Gonococci- positive	Gonococci- negative	Total
Within 8 days	2	5	7 (15.2%)	43	126	169 (61.9%)
" 8-30 "	6	21	27 (58.7%)	21	67	88 (32.2%)
More than 30 "	7	5	12 (26.1%)	9	7	16* (5.9%)
<i>Total</i>	15	31	46	73	200	273

* Including relapses after an apparent clinical cure.

The significant features emerging from this series are:

(1) In contrast to the 15.2% of cases that recovered within 8 days under the classical methods of local therapy, sulphapyridine gave no less than 61.9% of recoveries. Admittedly the control series is small, but it does reflect the general experience of the older observers.

(2) Sulphapyridine proved effective against non-gonococcal ophthalmia neonatorum as readily as against ophthalmia due to the gonococcus—in fact, there was a rather higher incidence of recovery within 8 days in the non-gonococcal group in contrast to the gonococcal group (63% against 58.9%).

(3) Staphylococci were present 111 times in the 200 cases of non-gonococcal ophthalmia neonatorum, and sulphapyridine proved effective not only against these organisms but also against a variety of other organisms, including various bacilli. Apart from the fact that there was no selective action on the gonococcus, it was clear that the whole range of causal organisms in ophthalmia neonatorum responded to sulphapyridine treatment.

(4) Investigation also showed that delay in starting sulphapyridine treatment did not influence its efficacy; 83 cases (63 non-gonococcal and 20 gonococcal) first received sulphapyridine after the infection had been established for more than 8 days; these cases responded as well as the less established infections.

(5) The optimum dosage of sulphapyridine was found to be: an initial dose of half a tablet (0.25 g.) followed by a quarter of a tablet (0.125 g.) four hourly day and night.

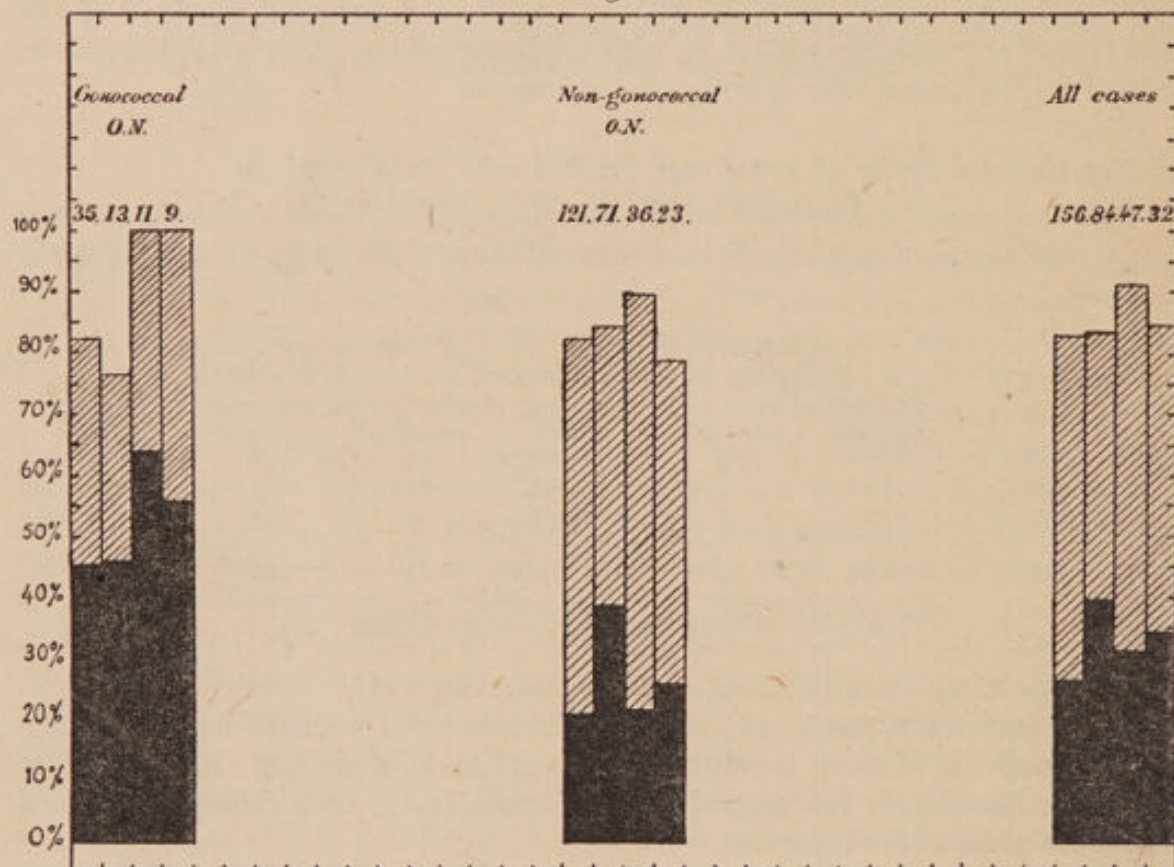
(6) Relapses were noted after apparent clinical cure in some cases, particularly

in the earlier ones, and intolerance to the drug was also observed, as shown by cyanosis and attacks of dyspnoea.

(2) *Series of 333 Cases treated with Standardized Doses of Various Sulphonamides*

The 333 cases of ophthalmia neonatorum treated during 1942-44 by the standard dosage elaborated towards the end of the first series gave distinctly better results than those obtained in the first series as a whole. In fact, a considerable percentage of cases showed clinical cure in 1-3 days; using cure within that period as the

Fig II



Comparative Efficacy of Various Sulphonamides in Ophthalmia Neonatorum.

The four columns in each block represent cases treated by sulphapyridine, sulphamezathine, sulphathiazole, and sulphadiazine respectively.

Heavy shading = percentage showing clinical cure within 1-3 days.

Light shading = percentage showing clinical cure within 4-8 days.

Numbers over each column = the total number concerned.

criterion of efficacy of these drugs, a comparative assessment of their value is possible. The data are set out in Table XIII, and the essential features are brought out in the following summary table (see also Fig. 11):

Comparative Value of some Sulphonamides

	Number of cases treated	Percentage cured within 1-3 days
Sulphapyridine	156	27.0
Sulphamezathine	84	39.3
Sulphathiazole	47	31.9
Sulphadiazine	32	34.4
Sulphanilamide	14	50.0

Sulphanilamide was employed in only a small group. Judging by the percentage of cases showing rapid cure it is not inferior to the other sulphonamides, but its clinical use led to considerable trouble in the way of toxic symptoms such as cyanosis and dyspnoea, and it was therefore discarded.

Sulphapyridine likewise showed considerably greater toxicity in comparison with the three remaining sulphonamides, though it was distinctly better tolerated than sulphanilamide. With sulphapyridine the optimum dose is not infrequently one that is not tolerated.

Experience with sulphamezathine, sulphathiazole, and sulphadiazine showed these three drugs to be well tolerated. Toxic symptoms have not been observed, and for the present it is difficult to indicate whether any one of the three is better than the others.

Non-Gonococcal Cases. This second series of 333 cases confirmed the finding of the earlier series that non-gonococcal cases respond to treatment. It seems, however, that they do so less rapidly than the cases due to the gonococcus. The subjoined summary extracted from Table XIII shows the actual data.

General Sulphonamide Therapy in 69 Gonococcal Cases and 264 Cases of Non-gonococcal Ophthalmia Neonatorum

Clinical cure	Gonococci-positive	Gonococci-negative
1-3 days	35 (50.7%)	73 (27.3%)
4-8 days	25 (36.3%)	146 (56.0%)
9-30 days: relapses, poor response and intolerance	9 (13.0%)	45 (17.2%)

It is seen that whilst 50.7% of gonococcal cases showed clinical cure in 1-3 days, only 27.3 of the non-gonococcal cases responded as rapidly. If the period of 1-8 days is taken the two groups give, respectively, 87% and 83.3% of clinical cures. Though the gonococcus responds most readily, it does not appear that any particular variety of non-gonococcal ophthalmia fails to respond. Particularly significant are the results obtained with the 34 cases in this series of 264 non-gonococcal cases of ophthalmia neonatorum showing inclusion bodies in scrapings of the conjunctival epithelium. No case proved completely resistant to treatment and the percentage distribution of cure within 1-3 days, 4-8 days, and 9-30 days, corresponds closely to that obtained for the non-gonococcal cases as a whole. This is seen from the subjoined table.

Results of Sulphonamide Treatment in 34 Cases of Ophthalmia Neonatorum showing Inclusion Bodies

Clinical cure	Number of cases	Percentage
1-3 days	9	26.4
4-8 days	21	61.8
9-30 days: relapses, poor response and intolerance	4	11.8

(3) *Factors influencing Sulphonamide Therapy*

Apart from the fact that cases of gonococcal ophthalmia neonatorum respond more readily than those due to other organisms, no other factor of material significance could be established as influencing sulphonamide therapy.

(a) *Delay in Starting Treatment.* The 333 cases treated by full doses of sulphonamides included 109 admitted after the infection had been established for 8 days or longer. There was no tangible difference in response of these cases compared to those admitted before the infection was established.

(b) *Degree of Severity.* The last 75 cases contained 26 in which the condition was mild, 34 in which it was moderate, and 15 severe, the criteria used being the amount and nature of discharge and the swelling of the lids. As can be seen from the sub-joined table, there was no striking difference in the percentage of cases giving a rapid cure in each of these subdivisions.

Clinical cure	Mild	Moderate	Severe
1-3 days	13	11	7
1-8 days	9	14	4
9-30 days: relapses, poor response and intolerance	4	9	4
	<hr/> 26	<hr/> 34	<hr/> 15

Whilst no fine conclusions can be drawn, it would appear that severe cases respond as well as those showing milder degrees of the infection. The rapid response that some severe cases give to sulphonamide treatment is so striking that the clinical impression was formed that severe cases do better than those less heavily affected. This is not borne out by the actual analysis. It is, however, clear that severe affections do not necessarily mean prolonged treatment.

(c) *Unilateral Cases.* No tangible difference in the results of treatment of the unilateral cases as contrasted with those in which both eyes were affected was observed.

(d) *Sulphonamide Resistant Cases.* In the second series of 333 cases just as in the first experimental series of 273 cases, some babies showed a poor response to the different sulphonamides. As can be seen from Table XIII, the 156 treated with sulphapyridine and the 84 with sulphamezathine by standard dosage showed no less than 17.3% and 16.7% of cases resistant to treatment. In so far as treatment was required for more than 8 days, or relapses and poor response were seen, or (in the case of sulphapyridine) intolerance, it should be stressed that an unsatisfactory response to sulphonamide does not generally mean complete failure; it is only exceptionally that the condition is not improved to a considerable extent, and only in rare instances is the response so poor that anxiety for the state of the eye and the ultimate outcome is felt. Characteristic of the findings amongst the sulphonamide resistant cases are the following data on 15 failures observed in the last 75 cases.

Of these 15 cases 7 showed poor response and 8 relapses. Of the 7 in the first group 5 were treated with sulphamezathine and the causal organisms were gonococcus in 2 cases, *Staphylococcus aureus* in 2 more, and diphtheroids in one. The 2 remaining cases were a case of staphylococcus with Koch-Weeks bacilli treated with sulphanilamide, and a case with non-haemolytic streptococcus treated with sulphathiazole. In these last two cases the general condition was not good; the one case suffered from severe bronchitis, and the other developed broncho-pneumonia whilst under treatment. Ultimately these cases cleared up either by the administration of another sulphonamide or by the local application of penicillin.

Seven of the eight relapses had been treated with sulphamezathine and the organisms present were: pneumococci and diphtheroids two cases each, gonococcus, no organisms, and inclusion bodies one case each. In one case, in which pneumococci were present, sulphanilamide had been used. In all these eight cases there was nothing in the general condition to explain the relapse, and they all cleared up when a further course of the original sulphonamide was instituted.

It must as yet be left an open question whether the sulphonamide resistant cases represent infections due to relatively resistant strains of organisms, or failures that would no longer be seen with a further increase in dosage.

(e) *Mode of Administration.* Local application of sodium sulphacetamide adequately buffered in concentrations of 10%, 20%, and 30% have been used in a series of twelve cases of ophthalmia neonatorum at various stages. It proved ineffective.

(4) *Complications*

In no case did any local complications arise during the course of treatment. Corneal haze was present in a number of babies on admission to the hospital, and this rapidly cleared with treatment. Twenty babies were admitted with corneal ulceration (all unilateral); in only three cases was the residual scar sufficiently dense to rouse fears as to the ultimate state of vision of the eye. All the other babies were discharged with corneae that were clinically clear. Vomiting, cyanosis, and dyspnoea, or other toxic symptoms, have not been observed with sulphamezathine, sulphathiazole, and sulphadiazine. No serious complications have been noted with the less tolerated sulphonamides (sulphapyridine and sulphanilamide).

(5) *Routine Management of Cases of Ophthalmia Neonatorum*

On admission a swab of the pus is taken, both for a smear preparation and for culture. The eyes are then irrigated with a bland lotion, such as half-normal saline solution at room temperature. Guttae atropinae sulphas 1% and drops of medicinal paraffin are instilled as a routine measure in all cases, and half a tablet of sulphamezathine (0.25 g.) crushed into powder is given by mouth in a teaspoonful of water or milk. Sulphamezathine administration is continued in doses of 0.125 g. every four hours, day and night, until forty-eight hours after a clinical cure is obtained. Local treatment consists of three-hourly irrigation with saline solution during the first day in cases with profuse discharge; as a rule there is no need for further irrigation on the subsequent days. After irrigation medicinal paraffin is instilled as a precaution against the sticking together of the lids. Atropine is instilled three times daily in cases with corneal haze or ulceration.

With this treatment swelling of the lids generally subsides within twelve hours after admission; purulent discharge disappears within twenty-four hours, so that a threatening purulent ophthalmia becomes a simple conjunctivitis giving no anxiety. The eyes are either dry or very nearly so within seventy-two hours.

4. PENICILLIN THERAPY

UNLIKE the sulphonamides, penicillin remains effective in the presence of pus. It therefore has possibilities for the local therapy of ophthalmia neonatorum as an alternative to general sulphonamide treatment. The use of penicillin as a local application in 85 cases of ophthalmia neonatorum has shown highly promising results when this agent is used in adequate concentration and at frequent intervals.

(1) *A Preliminary Series of 25 Cases treated with Penicillin in a Concentration of 500, 1,000 and 1,500 Units per Cubic Centimetre*

In this series 8 cases had been treated by drops in a concentration of 500 units per cubic centimetre, 7 with drops double that concentration and 10 with drops in a concentration of 1,500 units per cubic centimetre. The first group of 8 cases received instillations of one drop at hourly intervals during the first 24 hours and at two-hourly intervals subsequently. Only 3 of these 8 cases were cured, whilst two more showed an initial recovery which, however, was not maintained. Of the

7 cases in the second group (treated with drops in a concentration of 1,000 units per cubic centimetre) the method of application in 3 cases was the same as in the first group, and in the remaining 4 cases the penicillin was instilled at half-hourly intervals for 24 hours and hourly subsequently. Of these 7 cases four showed clinical cure, whilst two more relapsed after an initial recovery. In the final 10 cases of this series (treated with penicillin in a concentration of 1,500 units per cubic centimetre) the drops were instilled at more frequent intervals: half-hourly during the first 24 hours and hourly subsequently. Of these 10 cases 6 responded well, and one more showed relapse after an initial recovery. Clinical cure was obtained in 2 days, 3 days, and 6 days in the 3 cases responding satisfactorily to 500 units per cubic centimetre; in 2, 4, 2 and 5 days respectively in the 4 cases treated successfully with 1,000 units per cubic centimetre; and in 18 hours in 1 case, in 2 days in 4 cases, and in 3 days in one case in the 6 cases treated successfully with a concentration of 1,500 units per cubic centimetre. The following summary table shows the results set out in relation to causal organisms:

Results of Penicillin Therapy in 25 Cases

+ = Successful treatment; - = Failure

Organisms	Concentration of Penicillin: Oxford Units per cubic centimetre					
	500		1,000		1,500	
	+	-	+	-	+	-
Gonococcus	2	2*	2	0	2	3*
Staphylococcus	1	1†	1	-	1	-
Diphtheroids	-	-	1	-	1	1
Staphylococcus and bacilli	-	1	-	1	1	-
Unidentified diplococcus	-	-	-	-	1	-
Virus: assumed from the presence of inclusion bodies	-	1	-	2‡	-	-
<i>Total</i>	3	5	4	3	6	4

* Including one case which relapsed after initial clinical cure.

† Relapsed after clinical cure in 6 days.

‡ Both initially clear after 3 days and 4 days respectively, when relapse occurred.

Taken as a whole, this group of 25 cases suggested that with increasing concentration better results could be obtained. In the subsequent cases concentration of 2,500 units per cubic centimetre was therefore adopted as the standard solution.

(2) *A Series of 60 Cases treated with Penicillin in a Concentration of 2,500 Units per Cubic Centimetre*

This series falls into three groups:

(a) Twenty-two infants were treated with penicillin in a concentration of 2,500 units per cubic centimetre, the drops being instilled half-hourly for the first 3 hours and then hourly for 24 hours, and 2-hourly subsequently. In all but one case there was an excellent clinical response, recovery in some instances occurring in a matter of hours, as can be seen from the following summary*:

Time taken in hours for a clinical cure	Number of cases	
3-24	6	(3, 7, 12, 12, 18 and 24 hours respectively)
27-43	7	(27, 36, 37, 40, 40, 40, and 43 hours respectively)
50-100	7	(50, 60, 70, 78, 100, 100 and 100 hours respectively)

* One case is omitted from this table, as the complication of corneal ulcer—present on admission—delayed a return to normal.

(b) Thirteen cases were treated with penicillin at intervals of one hour for 6 hours, then 2-hourly for 24 hours, and subsequently at 3-hourly intervals. All these cases responded well, but one relapsed after 24 hours and another after 5 days. The time taken for a clinical cure ranged from 4 to 60 hours, the actual figures being 4 hours in 2 cases and 18, 20, 26, 30, 34, 38, 48, 56 and 60 hours in the others. The two cases that relapsed had initially taken 30 and 60 hours respectively to show clinical cure.

(c) In the final 25 cases in this series penicillin was applied intensively—at intervals of 5 minutes in the first instance. This was continued until all tendency to discharge ceased, when penicillin was applied half-hourly and so continued until the eye was dry and free from swelling, i.e. was clinically cured. Treatment was then maintained at hourly intervals for 12 hours, and at 2-hourly intervals for a further 24 hours to consolidate the clinical cure. In this group two cases showed a distinctly poor response, and of the remaining 23 cases 5 relapsed after an initial clinical cure. The results in the residual 18 cases were excellent, the time taken for clinical cure ranging from 25 minutes to 38 hours with an average of just under 10 hours. The following summary table shows the essential features:

Time taken in hours for a clinical cure	Number of cases	Time taken in hours for a clinical cure	Number of cases
Up to 1	1	12-16	1
1-4	4	16-20	2
4-8	5	21	1
8-12	3	38	1

The 5 cases that relapsed had initially shown clinical cure in 3, 11½, 16½, 22 and 33 hours respectively; the 2 cases that had failed to respond had been treated for 40 and 72 hours respectively.

As far as the time taken for clinical cure is concerned, these 18 successful cases (out of 25) compare favourably with the 20 comparable cases (out of 22) and the 11 (out of 13 cases) treated by the same concentration of penicillin but less intensively. The following comparative figures show this clearly:

Penicillin Drops in Concentration of 2,500 Units

Time taken in hours for clinical cure	Initial Treatment		
	At 5-minute intervals	At half-hourly intervals	At hourly intervals
	Number of cases		
Within 12	13	2	2
12-24	4	4	2
25-48	1	7	4
49-72	—	3	3
Over 72	—	4	—
	18	20	11

Average time per case: 10, 44 and 33 hours in the three series respectively.

It would, therefore, appear that intensive penicillin therapy (at 5-minute intervals) reduces the time taken for a clinical cure to one-third or one-quarter of that required when this drug is applied at hourly or half-hourly intervals.

Rapidity of Clinical Cure in Relation to Severity of the Affection

No fine distinctions can be drawn from the limited material so far available as to any difference in response by heavy and less heavy infections. There is nothing to suggest that clinically severe cases respond less readily. In fact, the troublesome cases practically all belonged to the group regarded as moderate; 2 of the 3 cases that gave no response to penicillin and 6 of the 7 cases that showed relapse were all initially cases of moderate severity. The following summary table brings out the salient features:

	Initial Treatment		
	At 5-minute intervals 23 cases	At half-hourly intervals 22 cases	At hourly intervals 13 cases
Mild	6 cases: clinical cure in 25 minutes to 21 hours. Average: 8 hours. No failures. No relapses.	5 cases: clinical cure in 7 to 78 hours. Average: 51 hours. No failures. No relapses.	3 cases: clinical cure in 4 to 56 hours. Average: 36 hours. No failures. No relapses.
Moderate	13 cases: clinical cure in 3 to 38 hours. Average (of 11 cases): 13 hours. Two failures. Four relapses.	11 cases: clinical cure in 3 to 100 hours. Average: 46 hours. No failures. No relapses.	5 cases: clinical cure in 30 to 60 hours. Average: 41 hours. No failures. Two relapses.
Severe	6 cases: clinical cure in 4 hours to 16½ hours. Average: 11 hours. No failures. One relapse.	6 cases: clinical cure in 36 hours. Average (of 5 cases): 37 hours. One failure. No relapses.	5 cases: clinical cure in 4 hours to 34 hours. Average: 16 hours. No failures. No relapses.

Efficacy in Relation to Causal Organisms

The following summary table of aetiological types brings out the time taken to achieve clinical cure in the successfully treated cases and shows the number of failures and relapses in each group.

	Initial Treatment		
	At 5-minute intervals 25 cases	At half-hourly intervals 22 cases	At hourly intervals 13 cases
Gonococcus	5 cases: 1½ to 10½ hours. Average (of 4 cases): 7½ hours. One failure. No relapses.	5 cases: 24 to 40 hours. Average (of 4 cases *): 35 hours. No failures. No relapses.	4 cases: 4 to 34 hours. Average: 21 hours. No failures. No relapses.
Staph. aureus	8 cases: 5¼ to 22 hours. Average: 10½ hours. No failures. Three relapses.	5 cases: 3 to 60 hours. Average: 20 hours. No failures. No relapses.	1 case: 4 hours. No failure. No relapse.
Staph. albus	—	4 cases: 27 to 43 hours. Average: 37 hours. No failures. No relapses.	—
Diphtheroids	3 cases: 3 to 3½ hours (in 2 cases). One failure. One relapse.	4 cases: 50 to 100 hours. Average (of 3 cases): 83 hours. One failure. No relapse.	1 case: 56 hours. No failure. No relapse.

	Initial Treatment		
	At 5-minute intervals 25 cases	At half-hourly intervals 22 cases	At hourly intervals 13 cases
Haemolytic streptococcus	1 case: 25 minutes. No failure. No relapse.	—	—
Gram negative diplococci	1 case: 33 hours. No failure. Relapse.	—	—
Friedlander's bacillus	1 case: 16 hours. No failure. No relapse.	—	—
Morax-Axenfeld bacillus	1 case: 16 hours. No failure. No relapse.	—	—
Gram positive cocci	—	—	1 case: 18 hours. No failure. No relapse.
No organisms nor inclusion bodies	1 case: 13½ hours. No failure. No relapse.	2 cases: 12 and 70 hours respectively.	4 cases: 48 to 60 hours. Average: 57 hours. No failures. One relapse.
Virus presumed from presence of inclusion bodies	4 cases: 3 to 38 hours. Average: 16 hours. No failures. No relapses.	2 cases: 78 and 100 hours respectively. Average: 89 hours. No failures. No relapses.	2 cases: 30 and 38 hours respectively. Average: — No failure. One relapse.

* In the fifth case return to normal was delayed owing to the presence of a corneal ulcer; it is therefore omitted from this calculation.

In this total of 60 cases there were, therefore, one failure in a series of 14 cases showing the gonococcus, and 2 failures in a series of 8 cases showing diphtheroids; there were no failures in the 14 cases due to the *Staphylococcus aureus* and the 4 showing *Staphylococcus albus*, whilst the single cases due to *Streptococcus haemolyticus*, Friedlander's bacillus, Morax-Axenfeld bacillus, gram negative diplococci, and gram positive cocci all responded. It would therefore appear that all the common causal organisms of ophthalmia neonatorum respond to penicillin therapy: no real significance can be attached to the three failures (1 gonococcus and 2 diphtheroids) in view of the satisfactory response in other (and more) cases of this type. It is also noteworthy that no failures were seen in the 7 cases in which no organism or inclusion bodies were present and in the 8 cases in which a virus appeared to be the responsible agent judging by the presence of epithelial inclusion bodies.

Whilst it is unwarranted to draw any conclusions as to greater clinical susceptibility of the different varieties of ophthalmia neonatorum to penicillin, it is clear that neither the occasional failures nor relapses are explicable in terms of bacterial morphology (Table XIV).

Mode of Use

For the present something like a 5-stage system is in use:

(1) On admission a swab is taken for smear and culture and the eye is irrigated with half-normal saline at room temperature. A drop of adrenalin 1:1,000 is instilled and a scraping is taken from the palpebral conjunctiva for examination for the presence of inclusion bodies. Gutt. Atropine Sulphas 1 per cent are instilled if the cornea is involved.

(2) Any pus that may have accumulated is wiped away with moist pledgets of cotton-wool and one drop of penicillin in a concentration of 2,500 units per cubic centimetre is instilled. The baby is now returned to its cot.

(3) The instillation of penicillin is continued every 5 minutes until there is no discharge. Irrigation is not needed as pus does not form to any extent; such thin mucoid discharge (with a yellowish tinge from the admixture of penicillin) as is present can be ignored; or if it clings to the lid margin it may be wiped away with moist pledgets of cotton-wool. Generally half-an-hour's to 3 hours' treatment (6 to 30 applications) is necessary before the eye is dry. This part of the treatment requires the full-time attention of the nurse. Swelling of the lids persists a little while after cessation of the discharge.

(4) When there is no longer any discharge, the instillation of penicillin is continued at half-hourly intervals. At this stage the conjunctiva and lids rapidly assume a normal appearance. When all swelling and tendency to moistness have disappeared—and this generally involves treatment for about 6 to 12 hours—penicillin therapy is continued to consolidate the clinical cure.

(5) Penicillin is now instilled hourly for 12 hours and 2-hourly for a further 24 hours.

Penicillin is well tolerated by the infant eye; occasionally a mild transitory flushing of the conjunctiva is observed. (In the present series no complications of any kind have been observed, but in a case treated subsequently to those recorded here, there was an instantaneous and marked swelling of the conjunctiva and lids on the instillation of penicillin in the form of a lamella; the swelling subsided within 12 hours, the child having been put in the meantime on sulphamezathine treatment.)

(3) *Comparison with General Sulphonamide Therapy*

It is likely that general sulphonamide therapy of ophthalmia neonatorum is already obsolete as a routine procedure. The results obtained by the intensive application of penicillin locally are almost as strikingly superior to those obtained by sulphonamide therapy as these in turn were over the classical methods. The reduction of the duration of treatment from weeks by the older methods to days by the sulphonamides is paralleled by the reduction of treatment from several days by the sulphonamides to as many hours by penicillin. Should it prove possible to standardize penicillin therapy to a less exacting routine than is required at present, an ideal method of treatment, free from the disadvantages of systemic medication, will indeed be achieved.

DISCUSSION AND CONCLUSIONS

THE past twenty years have seen a gratifying decline in the incidence of impaired vision and of blindness arising from ophthalmia neonatorum. This is suggested by the consistent trend shown in lowered incidence of impaired vision and blindness in the official returns of notification, and is confirmed by the returns made from homes for blind babies and from schools for blind children. The decline in blindness from ophthalmia neonatorum has been a considerable, though by no means the only, factor in decline in the incidence of blindness from all causes in childhood. There is evidence suggesting that this process has become greatly accelerated during the past few years; the introduction of sulphonamide therapy may well be responsible for this.

In contrast, there is no valid reason for believing that the incidence of ophthalmia neonatorum—as distinct from the complications of the affection—has declined to any extent. The notification rates would suggest a fairly steady, though by no means marked, decline over the past twenty-five years, but the notification rates cannot be accepted as a true indication of the incidence of the disease; the wide differences reported by different authorities would in itself rule this out. It would appear that these different rates reflect differences in concepts as to definition and role of notification in the treatment of the affection rather than actual differences in incidence. There is some reason for believing that, whilst notification and the machinery instituted for treatment have been brilliantly successful—though unevenly in different parts of the country—in eradicating the complications of ophthalmia neonatorum, the general public health measures and advances of medicine have failed substantially in lowering the incidence of the affection itself. It is likely that ophthalmia neonatorum is no less frequent to-day than it was twenty-five years and, possibly, forty years ago.

At least two causes have contributed to this unsatisfactory position. In the first place, the widespread belief that ophthalmia neonatorum is entirely or practically synonymous with gonococcal ophthalmia has tended to the comparative neglect of prophylactic treatment of maternal infections other than gonorrhoea. In view of the fact that gonococcal infection is no longer the main factor in the causation of ophthalmia neonatorum, it is obvious that attention requires to be given to other forms of maternal infection. The recognition of a virus type of ophthalmia neonatorum and the causative virus cervicitis only emphasizes how broadened the conception of maternal infection has become: the gonococcus, other bacteria, and the viruses all have to be considered.

Too much reliance on disinfection of the new-born baby's conjunctival sac is probably an equally significant factor in the persistence of a high incidence of ophthalmia neonatorum. The mechanical component of the Credé procedure—toilet of the unopened lids—is of course highly valuable in any type of possible infection, but there are theoretical reasons for doubting the efficacy of the chemical component of the procedure—the attempted disinfection of the conjunctiva by silver nitrate or its substitutes—against organisms other than gonococcus. This is of considerable importance in view of the low incidence of gonococcal infection in ophthalmia neonatorum to-day. It would therefore appear that, if ophthalmia neonatorum is to be eradicated, there is need not only for a clear recognition of its composite aetiology but also for a strict obstetric asepsis which includes intensive ante-natal care. The Credé procedure must be viewed as the last—and possibly the least—of a sequence of measures, and there is still much to be done to define the use and limitations of its chemical, as distinct from its mechanical, component. Silver nitrate may well be the best antiseptic, but there is some reason for believing that the organic silver substitutes are as effective and distinctly safer and less irritant.

The excellent results of sulphonamide and of penicillin therapy in ophthalmia neonatorum should eliminate much of the blindness that afflicted past generations. Further refinements in the chemotherapy of ophthalmia neonatorum are likely to develop, but the more urgent task to-day is for an obstetric art that has advanced beyond Credé. The immense reduction in the incidence of ophthalmia neonatorum that resulted from the work of Credé should not blind one to the fact that Gibson's advocacy "First, to remove, if possible, the disease in the mother during pregnancy" is still largely unachieved—and still remains the foremost aim if ophthalmia neonatorum is to be entirely eradicated. There is still much to be learnt about the

maternal infections that may lead to ophthalmia in the new-born, and there is still much to be done before adequate ante-natal services are widely available.

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TABLE I

*Notifications of Ophthalmia Neonatorum in
England and Wales, 1914-43*

	Number of births	Number of notified cases of ophthalmia neonatorum	Rate per 1,000 births
1914	879,096	6,166 (39 weeks)	
1915	814,614	6,806	8.34
1916	785,520	7,613	9.69
1917	668,346	6,716	10.05
1918	662,661	6,532	9.85
1919	692,438	8,648	12.49
1920	957,782	10,304	10.76
1921	848,814	8,313	9.79
1922	780,124	7,107	9.11
1923	758,131	6,592	8.70
1924	729,933	6,267	8.59
1925	710,582	5,748	8.09
1926	694,563	5,896	8.49
1927	654,172	5,891	9.01
1928	660,267	5,609	8.50
1929	643,673	5,448	8.46
1930	648,811	5,481	8.45
1931	632,081	5,158	8.16
1932	613,972	4,730	7.70
1933	580,413	4,056	6.99
1934	597,642	4,487*	7.51
1935	598,756	4,369*	7.30
1936	605,292	4,586*	7.58
1937	610,557	5,050*	8.27
1938	621,204	5,168*	8.32
1939	619,352	4,594*	7.42
1940	607,029 †	4,390*	7.23
1941	587,228 †	4,195*	7.14
1942	654,039 †	4,516*	6.90
1943	683,213 †	4,502*	6.59

† Provisional.

* These figures exceed those shown by the Ministry of Health in Table IV.

Source: Registrar-General's [Annual] Statistical Review[s].

TABLE II

Incidence of Ophthalmia Neonatorum in England and Wales, 1918-38
Case Rates per 1,000 Births recorded in Different Areas

Year	Adminis- trative Counties	County Boroughs	Metro- politan Boroughs	Total	Birming- ham	Leeds	Liverpool	Man- chester
1918	6.0	16.4	8.7	9.9	13.3	11.8	34.4	30.1
1919	8.1	19.9	10.6	12.5	16.3	13.2	36.4	35.6
1920	6.4	17.6	9.9	10.7	18.2	12.6	30.8	36.4
1921	5.6	15.8	10.8	9.8	19.6	10.5	30.1	33.6
1922	5.2	14.9	9.2	9.1	24.5	7.1	30.3	26.0
1923	4.9	14.6	8.3	8.7	22.8	11.5	34.2	23.2
1924	4.7	14.5	8.6	8.6	22.8	8.3	33.8	24.2
1925	4.5	13.5	8.6	8.9	18.4	6.0	35.9	23.1
1926	5.3	13.3	9.0	8.5	22.3	9.0	32.5	17.5
1927	5.8	13.2	11.1	9.0	23.4	11.0	32.9	14.9
1928	5.4	12.8	10.2	8.6	30.0	8.6	28.5	15.2
1929	5.3	12.7	10.4	8.5	30.7	5.0	30.8	11.6
1930	5.3	12.9	8.8	8.3	34.3	6.5	32.3	11.6
1931	5.1	12.6	9.6	8.2	35.9	7.3	38.4	10.2
1932	5.0	11.0	11.0	7.7	19.5	6.6	36.6	9.4
1933	4.8	9.8	9.3	7.0	11.6	6.6	35.1	9.4
1934	4.6	12.0	8.2	7.5	35.3	7.5	39.5	10.8
1935	4.2	12.5	6.9	7.3	41.5	7.9	38.6	12.1
1936	4.1	13.0	8.3	7.6	50.4	8.3	39.8	11.0
1937	4.7	14.3	8.3	8.3	56.8	5.8	46.9	10.7
1938	4.5	14.6	9.0	8.3	63.5	4.2	47.4	12.4

Source: Annual Reports of the Chief Medical Officer of the Ministry of Health.

TABLE III

*Incidence of Impaired Vision and Blindness from Ophthalmia Neonatorum
in some Areas of England and Wales, 1924-33*

1. *Administrative County of London*

Year	Births	Notifications		Vision Unim- paired	Vision Impaired*		Total Blindness*		Deaths (Various Causes)*	
		No.	Rate per 1,000	No.	No.	Rate per 100,000	No.	Rate per 100,000	No.	Rate per 100,000
1924	85,147	738	8.7	639	8	10.2	1	1.3	7	8.9
1925	82,401	714	8.7	634	10	13.0	2	2.6	12	15.6
1926	78,825	718	9.1	634	11	15.0	—	—	9	12.3
1927	73,263	815	11.1	739	6	8.8	—	—	10	14.6
1928	72,352	739	10.2	720	6	8.3	—	—	14	19.3
1929	70,089	726	10.4	699	13	18.5	2	2.9	8	11.4
1930	69,447	607	8.7	587	6	8.6	—	—	6	8.6
1931	65,684	625	9.5	611	7	10.7	—	—	5	7.6
1932	62,233	690	11.1	661	5	8.0	—	—	13	20.9
1933	56,743	526	9.3	501	4	7.0	2	3.5	12	21.1
Total	716,184	6,898	9.6	6,425	76	11.0	7	1.0	96	13.8

* Excluding Chelsea for 1924 and Lambeth for 1924-27. Rates adjusted accordingly.

Source: Returns specially obtained by the London County Council from the Medical Officers of Health of the Metropolitan Boroughs.

TABLE III (continued)

*Incidence of Impaired Vision and Blindness from Ophthalmia Neonatorum
in some Areas of England and Wales, 1924-33*

2. English County Boroughs

Year	Births	Notifications		Vision Unim- paired	Vision Impaired		Total Blindness		Deaths (Various Causes)*	
		No.	Rate per 1,000	No.	No.	Rate per 100,000	No.	Rate per 100,000	No.	Rate per 100,000
1924	223,182 (a)	3,289	14.7	3,165	54	24.2	9	4.0	50	22.4
1925	224,597 (b)	3,079	13.7	2,956	47	20.9	5	2.2	54	24.0
1926	224,452 (c)	3,041	13.5	2,637	48	18.2	7	3.1	71	31.6
1927	217,216 (d)	2,897	13.3	2,788	42	19.3	4	1.8	59	27.2
1928	218,772 (e)	2,855	13.0	2,733	51	23.3	9	4.1	57	26.1
1929	214,833 (e)	2,738	12.7	2,637	34	15.8	4	1.9	49	22.8
1930	215,973 (f)	2,856	13.2	2,755	42	19.4	2	0.9	46	21.3
1931	199,204 (f)	2,681	13.5	2,609	29	14.6	3	1.5	37	20.3
1932	192,494 (f)	2,270	11.8	2,215	19	9.9	3	1.6	36	20.4
1933	179,608 (g)	1,860	10.4	1,793	28	15.6	—	—	30	18.2
Total	2,110,331	27,566	13.1	26,288	394	18.7	46	2.2	489	23.2

(a) Excludes Birkenhead, Dudley, Preston, Portsmouth, Walsall, Sunderland, Oxford.

(b) Excludes Preston, Dudley, Portsmouth, Walsall, Oxford.

(c) Excludes Portsmouth, Dudley, Oxford.

(d) Excludes Dudley, Oxford, Croydon.

(e) Excludes Dudley, Oxford.

(f) Excludes Dudley.

(g) Excludes Dudley, Derby.

* Excludes Birmingham. Death rates amended accordingly.

Source: Information supplied by the Ministry of Health.

*3. Welsh County Boroughs **

Year	Births	Notifications		Vision Unim- paired	Vision Impaired		Total Blindness		Deaths (Various Causes)	
		No.	Rate per 1,000	No.	No.	Rate per 100,000	No.	Rate per 100,000	No.	Rate per 100,000
1924	12,083	145	12.0	138	3	24.8	1	8.3	2	16.5
1925	11,618	121	10.4	114	2	17.2	—	—	3	25.8
1926	11,156	134	12.0	122	1	9.0	1	9.0	8	71.7
1927	10,041	103	10.3	94	3	29.9	—	—	6	59.8
1928	10,015	88	8.8	77	1	10.0	—	—	10	99.8
1929	9,660	91	9.4	85	1	10.3	—	—	3	31.1
1930	9,586	77	8.0	74	—	—	—	—	2	20.9
1931	9,287	69	7.4	60	2	21.5	1	10.8	4	43.1
1932	8,757	53	6.0	48	—	—	1	11.4	4	45.7
1933	8,537	51	6.0	47	3	35.1	—	—	1	11.7
Total	100,740	932	9.25	859	16	15.9	4	4.0	43	42.7

* Cardiff, Merthyr Tydfil, Newport (Mon.) and Swansea.

Source: Information supplied by the Ministry of Health.

TABLE IV

*Incidence of Impaired Vision and Blindness from Ophthalmia Neonatorum in England and Wales, 1934-43**1. London County Council*

Year	Births	Notifications	Vision Unimpaired	Vision Impaired	Total Blindness	Under Treatment	Died	Removed District	Other Classifications
		No.	No.	Rate per 100,000	No.	Rate per 100,000			
1934	56,853	468	425	1.8	—	—	10	23	3
1935	55,780	380	331	—	—	—	10	28	—
1936	56,273	458	407	1.8	1	1.8	10	26	—
1937	55,011	456	402	3.6	1	1.8	9	30	—
1938	54,495	497	433	3.6	—	—	13	38	—
1939	52,366	403	375	1.9	—	—	5	17	—
1940	46,213†	308	282	—	—	—	—	24	—
1941	33,944†	197	180	—	—	—	2	13	—
1942	40,654†	208	192	2.4	—	—	6	9	—
1943	45,030†	238	221	—	—	—	8	4	—
1934-43	496,619	3,613	8	1.6	2	0.4			

† Provisional.

2. English County Boroughs

Year	Births	Notifications	Vision Unimpaired	Vision Impaired	Total Blindness	Under Treatment	Died	Removed District	Other Classifications
		No.	No.	Rate per 100,000	No.	Rate per 100,000			
1934	198,523	1,923	1,753	6.0	2	1.0	95	23	2
1935	198,123	2,037	1,854	6.0	3	1.5	89	38	7
1936	198,696	2,125	1,935	7.0	3	1.5	98	24	7
1937	199,219	2,400	2,143	9.0	2	1.0	107	73	13
1938	201,409	2,504	2,295	6.4	5	2.5	86	39	30
1939	198,625	2,198	1,990	5.5	1	1.0	126	44	4
1940	193,534†	2,158	1,985	2.1	1	0.5	108	42	—
1941	180,806†	1,969	1,720	1.6	—	—	181	43	1
1942	197,195†	2,308	2,074	—	—	—	159	54	—
1943	210,324†	2,279	2,114	1.4	1	0.5	85	33	—
1934-43	1,976,454	21,901	90	4.5	18	0.9			

D*

† Provisional.

TABLE IV (continued)
Incidence of Impaired Vision and Blindness from Ophthalmia Neonatorum in England and Wales, 1934-43
3. English County Councils and County Districts

Year	Births	Notifications	Vision Unimpaired	Vision Impaired	Total Blindness	Under Treatment	Died	Removed District	Other Classifications
		No.	No.	Rate per 100,000	No.	Rate per 100,000			
1934		(a) 824 (b) 597 (c) 1,421	764		3		13	15	—
1935	302,310	47	537 1,301 693	5.0	4	1.3	24 37 23	11 26 11	—
1936	305,666	4.2	490 1,183 717	5.6	1	0.3	9 32 10	9 20 15	—
1937	312,414	4.2	488 1,205 860	5.4	2 3 2	1.0	11 21 31	6 21 11	—
1938	319,152	4.8	534 1,394 832	3.4	1 3 —	0.9	9 40 26	8 19 23	—
1939	327,675	4.7	575 1,407 728	2.4	2 2 —	0.6	13 39 21	11 34 16	9
1940	330,959	4.0	461 1,189 669	3.0	1 1 2	0.3	11 32 8	6 22 15	—
1941	327,963 †	4.1	421 1,090 759	0.9	3 3 —	0.9	1 9 14	3 18 13	3
1942	331,916 †	3.9	446 1,205 739	1.2	—	—	14 28 11	7 20 12	13
1943	372,090 †	3.5	465 1,204 656	0.8	—	—	9 20 4	6 18 13	2
	384,623 †	3.2	471 1,127	1.3	1 1	0.2	14 18	6 19	3 4 7
1934-43	3,314,768	4.0	93	2.8	18	0.5			

(a) County Councils, excluding London County Council; (b) County Districts; (c) Total.

† Provisional

TABLE IV (continued)

Incidence of Impaired Vision and Blindness from Ophthalmia Neonatorum in England and Wales, 1934-43

4. *Wales*

Year	Births	Notifications		Vision Unimpaired		Vision Impaired		Total Blindness		Under Treatment	Died	Removed District	Other Classifications
		No.	Rate per 1,000	No.	No.	Rate per 100,000	No.	Rate per 100,000					
1934	39,956	226	5.6	211	2	5.0	—	—	7	3	3	—	
1935	39,187	229	5.8	197	7	17.9	7.6	7.6	3	7	4	8	
1936	37,909	197	5.2	178	4	10.5	2.6	2.6	7	5	2	—	
1937	37,175	223	6.0	211	—	—	2.7	2.7	1	6	3	1	
1938	37,625	217	5.8	201	1	2.6	8.0	8.0	2	7	3	—	
1939	37,402	209	5.6	198	2	5.3	—	—	4	4	1	—	
1940	39,319	245	6.2	222	2	5.1	—	—	5	5	10	—	
1941	40,562	248	6.1	223	3	7.4	2.5	2.5	11	4	6	1	
1942	44,100	238	5.4	226	—	—	—	—	5	5	2	—	
1943	43,236	188	4.3	174	—	—	—	—	—	2	7	—	
1934-43	396,471	2,220	5.6	21	21	5.3	2.3	2.3	—	—	—	5	

† Provisional.

5. *England and Wales as a whole*

Year	Births	Notifications	Vision Unimpaired	Vision Impaired	Total Blindness	Under Treatment	Died	Removed District	Other Classifications
				No.	Rate per 100,000	No.	Rate per 100,000		
1934	597,642	4,038	3,690	30	5.0	6	1.0	87	5
1935	598,756	3,934	3,565	36	6.0	7	1.2	105	15
1936	605,292	4,093	3,725	36	5.9	8	1.3	97	8
1937	610,557	4,599	4,150	31	5.1	7	1.2	158	15
1938	621,204	4,752	4,336	24	3.9	10	1.6	144	40
1939	619,352	4,134	3,752	24	3.9	2	0.3	129	7
1940	607,029	3,860	3,579	9	1.5	4	0.7	99	4
1941	587,228	3,713	3,328	10	1.7	1	0.2	91	14
1942	654,039	4,043	3,696	4	0.6	—	—	107	2
1943	683,213	3,996	3,636	8	1.2	2	0.3	118	12
1934-43	6,184,312	41,162		212	3.4	47	0.8		

† Provisional.

TABLE IV (continued)

Incidence of Impaired Vision and Blindness from Ophthalmia Neonatorum in England and Wales, 1934-43

6. *England by Counties, County Boroughs and County Districts and Wales as a whole, 1934-43*

	<i>Births</i>	<i>Notifica- cations</i>	<i>Vision Impaired</i>	<i>Rate per 100,000</i>	<i>Total Blind- ness</i>	<i>Rate per 100,000</i>
County Councils (excluding L.C.C.)	3,314,768	8,034	67	2.8	8	0.5
County Districts		5,344	26		10	
County Boroughs	1,976,454	21,901	90	4.5	18	0.9
L.C.C. and Metro- politan Boroughs	496,619	3,613	8	1.6	2	0.4
Wales	396,471	2,220	21	5.3	9	2.3
<i>Totals</i>	6,184,312	41,112	212	3.4	47	0.8
1934-40						
County Councils (excluding L.C.C.)	2,226,139	5,725	59	3.6	8	0.8
County Districts		3,824	22		9	
County Boroughs	1,388,129	15,345	84	6.0	17	1.2
L.C.C. and Metro- politan Boroughs	376,991	2,970	7	1.9	2	0.5
Wales	268,573	1,546	18	6.7	8	3.0
<i>Totals</i>	4,259,832	29,410	190	4.5	44	1.0
1941-43						
County Councils (excluding L.C.C.)	1,088,629	2,309	8	1.1	-	0.1
County Districts		1,520	4		1	
County Boroughs	588,325	6,556	6	1.0	1	0.2
L.C.C. and Metro- politan Boroughs	119,628	643	1	0.8	-	-
Wales	127,898	674	3	2.3	1	0.8
<i>Totals</i>	1,924,480	11,702	22	1.1	3	0.1

Source: Information supplied by the Ministry of Health.

(Impaired vision or blindness developing in cases noted *Under Treatment* in sections 1-5 of this Table appear under the appropriate column in the succeeding year.)

TABLE V

Ophthalmia Neonatorum: Case-rate and Incidence of Impaired Vision and Blindness in Birmingham, 1924-43

Year	Births	Notifications		Vision Impaired		Total Blindness	
		No.	Rate per 1,000	No.	Rate per 100,000	No.	Rate per 100,000
1924	18,329	413	22.5	2	10.9	1	5.4
1925	17,858	335	18.8	3	16.8	2	11.2
1926	17,859	395	22.1	3	16.8	—	—
1927	17,447	409	23.4	2	11.5	—	—
1928	17,248	530	30.7	14	81.1	4	23.1
1929	16,980	522	30.7	5	29.4	1	5.9
1930	17,211	596	34.6	6	34.9	—	—
1931	17,313	617	35.6	3	17.3	—	—
1932	16,431	319	19.4	3	18.2	2	12.1
1933	15,151	176	11.6	1	6.6	—	—
Total	171,827	4,312	24.0	42	24.4	10	5.8
1934	15,757	556	35.3	1	6.4	—	—
1935	15,897	658	41.3	—	—	—	—
1936	16,300	812	49.5	1	6.1	—	—
1937	16,849	962	57.1	—	—	—	—
1938	17,425	1,105	63.4	1	—	—	—
1939	17,461	1,001	57.3	—	—	—	—
1940	17,213	1,036	60.2	—	—	—	—
1941	15,961	928	58.1	—	—	—	—
1942	18,664	1,203	64.4	—	—	—	—
1943	21,157	1,165	55.0	—	—	—	—
Total	172,684	9,426	54.6	3	1.7	—	—

Source : Registrar-General's [Annual] Statistical Reviews and information supplied by the Ministry of Health.

TABLE VI

Ophthalmia Neonatorum—Scotland, 1914-42
Case-rate and Incidence of Impaired Vision and Blindness

Year	Births	Notifications		Vision Impaired		Total Blindness	
		No.	Rate per 1,000	No.	Rate per 100,000	No.	Rate per 100,000
1914	123,934	607	4.9				
1915	114,181	615	5.4				
1916	109,942	768	7.0				
1917	97,441	829	8.5				
1918	98,554	844	8.6				
1919	106,268	1,431	13.5				
1920	136,535	1,871	13.7				
1921	123,201	1,897	15.4				
1922	115,082	1,485	12.9	2	1.7	3	2.6
1923	111,902	1,390	12.4	6	5.4	—	—
1924	106,900	1,289	12.0	12 *	11.2	3	2.8
1925	104,137	1,246	12.0	2 †	1.9	1 *	1.0
1926	102,449	1,295	12.6	2	2.0	—	—
1927	96,672	1,311	13.6	12 *	12.4	—	—
1928	96,822	1,292	13.3	3	3.1	2	2.1
1929	92,880	1,215	13.1	12 †	12.9	—	—
1930	94,549	1,431	15.1	4 †	4.2	1	1.1
1931	92,220	1,443	15.6	5	5.4	1	1.1
1932	91,000	1,662	18.3	3	3.3	1	1.1
1933	86,546	1,472	17.0	7 *	8.1	—	—
1934	88,836	1,481	16.7	5	5.6	—	—
1935	87,928	1,345	15.3	4	4.5	1	1.1
1936	88,928	1,379	15.5	8	9.0	1	1.1
1937	87,810	1,732	19.7	2	2.3	1	1.1
1938	88,627	1,705	19.2	3	3.4	—	—
1939	86,899	1,328	15.3	—	—	—	—
1940	86,403	1,152	13.3	1	1.2	—	—
1941	89,743	908	10.1	—	—	—	—
1942	90,694	1,023	11.3	1	1.1	—	—
1943	94,686	1,019	10.7	1	1.1	—	—
1922-24	333,888	4,164	12.5	20 *	6.0	6	1.8
1924-33	964,179	13,656	14.0	62 †	6.4	9 *	0.9
1934-43	890,554	13,072	14.6	25	2.8	3	0.4
1934-40	615,431	10,122	16.4	23	3.7	3	0.5
1941-43	275,123	2,950	10.7	2	0.7	—	—

* Including 1 died.

† " 2 "

‡ " 9 "

Source: Annual Reports of the Department of Health for Scotland and information supplied by the Department.

TABLE VII

See pp. 58 and 59.

TABLE VIII
Causes of Blindness in Children up to the Age of 16 at 7 Provincial Blind Schools, 1944

	Birmingham Royal Institution for the Blind		East Anglian Schools for the Blind		Royal Normal College		Royal School for the Blind, Bristol		Royal Victoria School for the Blind		Swiss Cottage		West of England Institution for the Blind		Total		Percentage
	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	
<i>Acquired:</i>																	
Ophthalmia Neonatorum	10	8	-	2	4	3	-	-	3	2	6	5	4 (2+2)	1	27	21	48
Interstitial Keratitis	1	-	2	-	-	-	-	-	-	1	-	-	-	-	4	1	5
Other Syphilitic Lesions	2	2	-	-	-	1	1	2	-	1	1	2	-	1	4	9	13
Phlyctenular Ophthalmia	1	-	-	-	-	-	-	-	-	-	-	-	-	-	1	1	2
Purulent Ophthalmia of later years	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Keratitis and Corneal Ulcer	1	1	2	-	-	-	-	-	1	-	-	-	-	-	4	1	5
Iridocyclitis	-	-	1	-	1	1	-	-	1	1	-	-	-	-	2	2	4
Choroiditis	-	-	-	-	-	-	-	-	1	2	-	1	-	-	1	3	4
Trauma	1	1	-	-	1	-	2	-	1	-	-	-	-	-	5	1	6
Sympathetic Ophthalmia	2	3	-	-	-	-	-	-	-	-	3	-	-	-	5	3	8
Birth Trauma	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	1
War Trauma	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Measles and other Infectious Fevers	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Meningitis	5	2	-	-	1	2	2	-	-	1	2	1	-	-	1	6	2
Cerebral Tumour	1	-	-	-	-	-	-	-	-	-	-	-	-	-	10	1	16
Optic Atrophy	4	3	5	1	-	-	2	-	-	-	-	-	-	-	2	-	2
Endophthalmitis	2	1	-	-	-	-	-	1	1	-	3	5	3	2	17	11	28
<i>Congenital:</i>																	
"Congenital Defects"	6	7	4	4	-	1	1	-	3	1	3	2	2	3	19	18	37
Anophthalmos	-	2	-	1	-	-	-	-	1	1	-	-	-	-	1	4	5
Microphthalmos	4	2	2	1	1	-	-	-	1	3	-	-	-	-	8	5	13
Buphthalmos	7	3	2	1	2	-	4	1	1	1	7	2	-	1	23	9	32
Colobomata	-	1	1	1	1	2	1	2	5	5	2	3	2	1	12	15	27
Nystagmus	8	2	-	1	1	-	3	-	1	2	5	2	-	6	20	13	33
Albinism	3	3	-	-	3	1	2	1	-	-	2	3	-	-	7	7	14
Retinitis Pigmentosa	-	1	-	-	-	-	1	-	-	-	1	2	-	-	5	3	8
Macular Dystrophy	1	1	-	-	-	-	-	-	-	-	-	-	-	-	3	1	4
Retinal and Choroidal Defect	1	1	-	-	-	1	-	-	-	-	1	-	-	-	3	1	4
Cataract	18	9	8	5	2	3	7	4	5	3	3	4	11	4	54	32	86
Subluxation of Lenses	3	5	-	-	-	-	-	-	1	1	-	-	-	-	5	6	11
Glioma	1	1	2	-	-	-	-	-	-	-	-	3	1	-	4	4	8
Detachment	2	-	-	-	-	-	-	-	-	-	-	-	-	-	5	-	5
Myopia	6	4	7	7	1	1	3	8	-	-	1	3	12	6	34	32	66
Undetermined	1	-	5	2	2	3	-	-	-	-	-	-	3	1	11	6	17
	92	62	45	25	20	19	39	19	25	27	42	38	44	27	307	217	524
																	99.7

Sources: Returns supplied by Ramsay Garden, Esq. D.O.M.S., Philip Jameson Evans, Esq. F.R.C.S., and the Head Masters of the Blind Schools concerned.

TABLE

Causes of Blindness in Infants up to 5 Years of Age

	1920	1921	1922	1923	1924	1925	1926	1927	1928	1929	1930	1931	1932	1933	1934
Infections:															
Ophthalmia Neonatorum	10	4	6	9	7	7	9	7	4	7	7	2	8	10	6
? Ophthalmia Neonatorum	-	1	3	-	-	-	-	-	1	-	1	-	-	-	-
Measles	1	1	-	1	-	2	2	3	3	4	-	1	-	3	-
Purulent Ophthalmia	-	1	-	-	2	1	-	-	1	2	1	-	1	-	1
Corneal Ulceration and Nebulae	-	-	-	1	-	-	-	2	-	-	2	1	-	-	1
Interstitial Keratitis	-	-	-	-	-	-	1	1	-	-	-	-	-	-	-
Disseminate Choroiditis	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Uveitis	-	-	-	-	-	-	2	-	1	-	-	-	-	-	-
Endophthalmitis	-	-	-	-	-	-	1	-	1	-	-	1	1	1	-
Panophthalmitis	-	-	-	-	-	-	-	-	-	-	-	2	-	-	-
Meningitis	-	-	-	1	1	-	-	1	1	-	-	-	-	1	-
Syphilis	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-
	12	7	9	12	11	10	15	14	12	13	11	7	10	15	8
Trauma:															
Accident	-	-	-	-	-	-	-	1	-	1	-	-	-	-	-
Obstetric Trauma	-	-	-	-	-	-	-	-	-	-	-	1	-	1	-
Sympathetic	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-
Enemy Action	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
	-	-	-	1	-	-	-	1	-	1	-	1	-	1	-
Congenital:															
"Congenital Anomalies"	-	-	1	5	5	6	2	3	3	11	4	3	3	3	2
Nystagmus	-	-	-	-	-	-	-	2	-	-	-	-	-	-	1
Microphthalmos	2	1	2	1	1	3	-	-	1	-	4	5	-	5	1
Anophthalmos	1	-	1	1	2	1	1	-	1	1	-	-	1	3	1
Buphthalmos	1	-	1	1	-	1	1	-	2	1	-	1	1	-	-
Absence of Optic Nerves	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-
Cataract	-	-	1	1	1	1	1	1	-	1	2	1	1	3	5
Albinism	1	-	-	1	1	-	-	-	-	-	-	-	-	-	-
Retinitis Pigmentosa	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-
Glioma	-	-	-	-	-	3	1	1	-	2	-	3	2	-	1
	5	1	6	10	10	15	7	7	7	16	11	13	8	14	11
Indefinite Aetiology:															
Optic Atrophy	-	-	-	1	1	1	1	-	-	1	-	-	4	-	-
Detached Retinae	-	-	-	-	1	-	-	-	-	-	-	-	-	-	1
"Tumours"	-	-	-	-	-	3	-	-	-	-	-	-	-	-	-
"Cerebral"	-	-	-	-	-	-	-	2	-	-	-	-	-	-	-
Keratomalacia	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-
	-	-	-	1	2	4	2	2	-	1	-	-	4	-	1
Not fully certified	1	1	-	3	1	13	1	1	2	-	3	1	4	1	1
Total															

Source: Information supplied by Mr. W. McG. Eagar and

TABLE IX
England and Wales, 1923-43: Total Blind Population in Age-Groups

Year	0-5	5-16	16-21	21-50	50-70	Over 70	Unknown	Total
1923	231	2,723	1,567	10,955	12,397	8,026	619	36,518
1925	257	2,720	1,682	12,200	15,048	9,865	368	42,140
1927	258	2,554	1,670	12,967	17,232	11,958	183	46,822
1929	258	2,438	1,623	13,901	20,149	14,304	54	52,727
1931	225	2,355	1,667	14,740	23,409	17,568	634	60,598
1933	244	2,089	1,613	14,955	25,066	19,277	164	63,408
1936	206	1,855	1,391	15,129	26,455	22,369	129	67,534

Year	0-1	1-5	5-16	16-21	21-40	40-50	50-65	65-70	Over 70	Unknown	Total
1938	11	184	1,676	1,350	7,939	7,278	18,904	9,247	25,141	147	71,875
1939	21	169	1,619	1,311	8,137	7,476	19,260	9,601	26,730	168	74,494
1940	9	186	1,499	1,327	7,964	7,589	19,020	9,860	26,811	153	74,418
1941	11	190	1,425	1,227	7,920	7,491	18,954	9,716	27,250	140	74,324
1942	9	192	1,369	1,124	7,728	7,393	18,493	9,784	29,053	161	75,306
1943†	13	204	1,355	1,078	7,602	7,203	18,354	9,629	30,884	185	76,507

† Provisional.

Source: Information supplied by the National Institute for the Blind and by the Southern Regional Association for the Blind based on returns by Local Authorities.

TABLE X
*Incidence of Blindness in Children aged 5-16 * years*
England and Wales, 1923-43

Year	Population aged 5-16 years	No. of Registered Blind aged 5-16 years	Rate per 100,000
1923	7,477,143	2,723	36.4
1925	7,306,761	2,720	37.2
1927	7,385,359	2,554	34.6
1929	7,262,590	2,438	33.6
1931	7,166,056	2,355	32.9
1933	7,246,446	2,089	28.8
1936	6,890,730	1,853	26.9
1938	6,694,300	1,676	25.0
1939	†	1,619	24.2 †
1940	†	1,499	22.4 †
1941	†	1,425	21.3 †
1942	†	1,369	20.5 †
1943	†	1,355	20.3 †

* i.e., between the ages 5-16 years.

† Population figures not available for publication.

‡ On 1938 population figures.

Source: Table IX supplemented by information supplied by the Registrar-General.

TABLE XI
Incidence of Blindness in Children aged 3-16 years
London County Council, 1910-43

Year	Number of Elementary School Children scheduled aged 3 and upwards *	Number of Children passed for Blind Schools			Rate per 100,000
		Boys	Girls	Total	
1910	888,987			118	13.27
1911	889,446			103	11.58
1912	892,359			106	11.88
1913	891,584			125	14.02
1914	893,848	57	59	116	12.98
1915	886,368			121	13.65
1916	878,887			60	6.83
1917	871,407			60	6.88
1918	863,926			126	14.58
1919	856,446			81	9.46
1920	854,979	37	44	81	9.46
1921	835,779	47	51	98	11.46
1922	816,578	47	41	88	10.53
1923	797,378	45	44	89	10.90
1924	801,131	27	32	59	7.40
1925	797,149	51	34	85	10.61
1926	782,005	34	36	70	8.78
1927	766,860	15	18	33	4.22
1928	742,388	14	21	35	4.56
1929	717,917	24	21	45	6.06
1930	701,461	12	10	22	3.06
1931	685,004	13	10	23	3.28
1932	677,841	10	8	18	2.63
1933	670,678	16	9	25	3.69
1934	635,470	4	8	12	1.79
1935	600,262	1	4	5	0.79
1936	571,937	9	5	14	2.33
1937	543,611	7	5	12	2.10
1938	520,847	5	4	9	1.66
		3	3	6	1.15
	22,332,533			1,764	7.89
1939	498,083	Not available			
1940 †	106,750	5	2	7	6.56
1941 †	183,000	13	4	17	9.29
1942 †	269,300	4	8	12	4.46
1943 †	235,000	6	3	9	3.83

* Including Special Schools, e.g., P.D., M.D., Open Air, Nursery and Hospital schools.

† Estimates.

Source: Annual Reports of the School Medical Officer of the London County Council supplemented by information from the Council's School Medical Service.

TABLE XII
Blindness and its Causes in School Children, 1905-44

	Harman*		L.C.C.*		Board of Education †		L.C.C. †		L.C.C. †		L.C.C. †		Provincial ††	
	No.	Per-cent- age	No.	Per-cent- age	No.	Per-cent- age	No.	Per-cent- age	No.	Per-cent- age	No.	Per-cent- age	No.	Per-cent- age
Ophthalmia Neonatorum	367	19.8	42	11.3	295	28.5	46	21.1	14	14	5	9.8	48	9.2
Purulent Conjunctivitis of later years	90	4.8	—	—	29	2.8	—	—	—	—	—	—	—	—
Phlyctenular Ophthalmia	66	3.5	30	8.1	34	3.3	12 §	5.5	3 §	3	—	—	1	0.2
Interstitial Keratitis and Iridocyclitis	413	22.3	59	16.0	81	7.8	20	9.2	1	1	1	2.0	26	5.0
Optic Atrophy, Chorioretinitis and Choroiditis	349	18.8	52	14.1	141	13.6	43	19.8	16	16	8	15.7	28	5.3
Injuries and/or Sympathetic Ophthalmia	29	1.6	5	1.5	21	2.0	3	1.4	4	4	3 ¶	5.8	17	3.2
Other defects	—	—	—	—	48	4.6	2	0.9	6	6	4	7.9	49	9.2
Congenital and Hereditary Lesions, including Myopia	541	29.1	181	49.0	387	37.3	92	42.2	56	56	30	58.8	355	67.6
	1,855		369		1,036		218		100		51		524	

* Based on the numbers passed as suitable for blind schools.

† " " Table VIII.

|| Interstitial keratitis, iridocyclitis, choroiditis and other syphilitic lesions.

† Based on the pupils attending blind schools.

§ Superficial keratitis.

¶ Including two due to fracture of skull.

Sources: Ministry of Health: Departmental Committee on the Causes and Prevention of Blindness. Final Report, 1922. London County Council: Annual Reports of School Medical Officer for 1927, 1931 and 1938, and information supplied by the Council's School Medical Service.

TABLE XIII

Comparative Results with some Sulphonamides in Ophthalmia Neonatorum

333 Cases treated with Full Doses

(0.25 g. on admission and 0.125 g. 4-hourly day and night until 48 hours after clinical cure)

Clinical Cure	Sulphapyridine			Sulphanilamide			Sulphamezathine			Sulphathiazole			Sulphadiazine		
	Gonococci-positive	Gonococci-negative	Total	Gonococci-positive	Gonococci-negative	Total	Gonococci-positive	Gonococci-negative	Total	Gonococci-positive	Gonococci-negative	Total	Gonococci-positive	Gonococci-negative	Total
1-3 days	16 (45.7%)	26 (21.5%)	42 (27.0%)	1	6	7 (50.0%)	6 (46.2%)	27 (38.0%)	30 (39.3%)	7 (63.6%)	8 (22.2%)	15 (31.9%)	5	6	11 (34.4%)
4-8 days	13 (37.1%)	74 (61.1%)	87 (55.7%)	-	3	3 (21.4%)	4 (30.7%)	33 (46.5%)	37 (44.0%)	4 (36.3%)	24 (66.6%)	28 (59.6%)	4	12	16 (50.0%)
9-30 days; relapses, poor response and intolerance	6 (17.1%)	21 (17.4%)	27 (17.3%)	-	4	4 (28.6%)	3 (23.1%)	11 (15.5%)	14 (16.7%)	-	4 (11.1%)	4 (8.5%)	-	5	5 (15.6%)
Totals	35	121	156	1	13	14	13	71	84	11	36	47	9	23	32

TABLE XIV

Summary showing the Results in 85 Cases of Ophthalmia Neonatorum treated by Various Concentrations of Penicillin

(+ = successful treatment; \pm = relapse after successful treatment; — = failure)

	Concentration of Penicillin: Oxford Units per cubic centimetre											
	500			1,000			1,500			2,500		
	+	±	—	+	±	—	+	±	—	+	±	—
Gonococcus	2	1	1	2	0	0	2	1	2	13	—	1
Staphylococci	1	—	1	1	—	—	1	—	—	15	3	—
Diphtheroids	—	—	—	1	—	—	1	—	1	5	1	2
Staphylococci and bacilli	—	1	—	—	—	1	1	—	—	—	—	—
Unidentified diplococci	—	—	—	—	—	—	1	—	—	1	1	—
Haemolytic streptococci	—	—	—	—	—	—	—	—	—	1	—	—
Friedlander's bacillus	—	—	—	—	—	—	—	—	—	1	—	—
Morax-Axenfeld bacillus	—	—	—	—	—	—	—	—	—	1	—	—
No organisms nor inclusion bodies found	—	—	—	—	—	—	—	—	—	6	1	—
Virus: assumed from the presence of inclusion bodies	—	—	1	—	2	—	—	—	—	7	1	—
	3	2	3	4	2	1	6	1	3	50	7	3

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