
COLLECTED ESSAYS ON THE
MORTALITY OF INFANTS

by

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I - INTRODUCTION.

One of the most startling and satisfactory phenomena which vital statistics have shown is the enormous decline within the present century of the mortality of infants and young children. Prior to 1901, some 15 per cent. of all infants born failed to celebrate their first birthday; but the last 25 years have seen this proportion steadily dwindling to one half of its original value. Relatively sudden in its commencement, showing (until possibly the last two or three years) no appreciable slackening in its rate of improvement, and coincident with energetic medical efforts of control on a national scale, this amelioration of the fate of infants is viewed by all, except a relatively small though influential sect of eugenists, with considerable gratification and as a triumph of hygiene greater than or at least equal to the conquest of typhus, enteric and other epidemic diseases. Careful examination of the causes of death to which the major part of the decline can be attributed offers but scanty support for the gloomy view that this saving of life, at present apparently beneficial, is an ultimate danger to the well-being of the race. There is not a shadow of evidence pointing to the present decline in mortality having selected for its operation the eugenically unfit. The unconscious compliment to the increase in our medical and surgical skill in saving or prolonging the life of the unfit, implicit in the acceptance of this view, is moreover quite undeserved. The more general view is that the improvements in infant and child mortality manifested in our vital statistical data represent a permanent benefit to the population; but concerning the precise factor, or the relative importance of various probable factors responsible for this change, divergence of

of opinion is immediately in evidence. Improvements in domestic and municipal sanitation, deliberate efforts of conservation of infant life and health by ante-natal, infant welfare and "toddler" clinics, improvements in the health of the population at reproductive ages, more adequate and more intelligent maternal care and concomitant reduction in the birth rate are some of the many factors to which the changes are attributed.

To extricate, if possible, from the many suggested causal agencies those which have been most influential is obviously a task of extreme practical importance. Only by so doing can the energy and money expended at present be directed to and concentrated in the channels offering the most fruitful returns.

The present researches have been directed to this end, and an attempt has been made to explain the causes of the recent decline in mortality and to inquire to what extent certain factors, supposedly influential to infant life, can account for the variations in infant mortality observed when the rates in different sections of the country are examined. Those studies are concerned mainly with the influence of external agencies acting on the infant. Up to the present, the only data to which I have had access and which deal with the changes taking place in the internal body mechanism during the early stages of life are a series of hospital autopsy records; and the part of these results which seem to have a direct bearing on the present problems has been included in this study.

The very nature of the task, when, as in the present instance, numerous factors are involved, necessitates the use of the statistical technique, but I have tried as far as possible to free the text from technical language, and, where this has not been wholly practicable, have inserted explanatory notes in a short appendix.

II. THE DECLINE IN INFANT MORTALITY AND ITS POSSIBLE CAUSES.

Although many measures which may influence the mortality of infants have within recent years come into operation, it is convenient and permissible to classify these into one or other of two broad categories. In the first place, many general measures intended to ameliorate the life and health of the people at all ages must be taken into consideration. Here we have such improvements as have been effected or attempted in the unhealthy conditions of urban life in particular, improved housing, improved domestic and municipal sanitation, control of the milk supply etc. The manner in which these may affect the health of infancy is obvious and requires no elaboration. In the second instance, we have to consider the effects of those measures which have been specially directed towards the infant and the pregnant or nursing mother. From a small beginning in private philanthropic work has now sprung a vast national machinery of maternity and child welfare, a scheme which resolves itself into component parts directed towards improvements in the conditions effecting the three special periods of infant life, the ante-natal, natal and post-natal. The aims and objects of ante-natal supervision may be summed up briefly. The scheme is designed (Fairbairn, 1925) to maintain the health of the mother during pregnancy, to prepare her for the nursing and general management of her infant after its birth, to attempt to preserve the pregnancy to full-time and, above all, to foresee and take steps to avoid the preventible difficulties in labour. Obviously work of this nature, efficiently carried out, should influence the death rates at each/

each of the three periods of infant life in a definite manner. It should (1) reduce the preventible sickness and mortality among the mothers themselves, (2) reduce the proportion of pregnancies which do not reach full-time, (3) reduce the number of deaths of infants and mothers from accidents of childbirth, and (4) in the post-natal period should result in a diminution in those causes of death which are mainly dependent on the lack of intelligent maternal care. Natal work resolves itself into the adequate provision of lying-in accommodation and skilled obstetrical assistance till the end of the puerperium. Its effects will be manifested by reduction of the death-rates of mothers and infants from diseases and accidents complicating labour. The post-natal or welfare clinic is mainly educative and advisory. For purposes of treatment, which does not form part of the usual routine of the clinic, special treatment clinics have in some instances been formed to work in co-operation with the welfare clinic; but in places where these have not been instituted many of the ordinary ailments of infancy can be treated at the welfare clinic and the more serious cases referred to hospital. The total effect of the maternity and child welfare movement, granted efficient co-ordination between its subdivisions, should be that more intelligent maternal care is bestowed on the infant and that diseases are treated at an earlier stage and therefore more effectively. Apart from all such deliberate efforts to conserve infant life, two great vital-statistical changes, namely the improvement in the health of persons of reproductive ages and the decline in the birth rate, deserve consideration because of the effects which they may have had on infant mortality.

Analysis/

Analysis of the Data.

The data to which we have confined our attention in the present instance are the rates of mortality in England and Wales of the years 1911-25 inclusive. The differences in the classification of the causes of death introduced in 1911 and the non-distribution of births and deaths prior to that date preclude for the most part comparisons over a longer period of time. Since the rates from the several causes of death vary so much in actual magnitude, to ensure comparability in the changes which have taken place in the different causes of death, index numbers have been calculated for each of the 15 years, taking as base line the mean rate for the whole period. Each annual death-rate from any cause is then expressed as a percentage of the average for that cause, and the regression coefficients* calculated for each series. In some instances the actual death-rates do not follow with any measure of exactitude the course indicated by the regression straight line; but since our object is to obtain a simple and at the same time reasonably accurate expression of a general tendency, any attempt to follow irregularities in the trend of mortality by more complicated curves would defeat the end in view. Diagram I, which shows for some of the main causes of infant mortality the actual course and that indicated by the straight line, brings out with what degree of accuracy the regression coefficient represents the changes which have taken place within the period under consideration. In view of the fact that the worst examples were chosen for diagrammatic representation, the results may, on the whole, be regarded with some satisfaction. In the tables which follow a negative coefficient represents a decreasing, a positive coefficient an increasing death-rate. The appended errors are standard errors/

* See Appendix.

TABLE 1.

Decline in Various Causes of Infant Mortality in
England and Wales (1911-25).

Cause of death	Regression coefficient
Tuberculosis (all forms)	- 7.90 \pm 0.73
Diarrhoea and enteritis	- 9.69 \pm 2.74
Infectious diseases	- 4.54 \pm 1.43
Measles	- 6.98 \pm 1.94
Whooping cough	- 3.24 \pm 1.65
Bronchitis and pneumonia	- 1.28 \pm 0.64
Abdominal tuberculosis	-11.73 \pm 1.06
Syphilis	- 2.55 \pm 1.24
Convulsions	- 6.52 \pm 0.36
Malformations	+ 0.98 \pm 0.28
Injury at birth	+ 2.21 \pm 0.41
Premature birth	- 0.66 \pm 0.27
Atrophy, debility and marasmus	- 6.52 \pm 0.39
Meningitis	- 7.15 \pm 0.70
Overlying	- 8.29 \pm 0.72
Infant mortality (all causes)	- 3.59 \pm 0.46

errors, so that as a rough criterion of the significance attaching to any change twice the standard error may be taken. If the coefficient does not exceed this standard, it may be inferred that there has been no definitely proven tendency for that death rate to rise or fall within the period under review.

(a) Post-natal mortality. Table 1 shows the rate of decline in infant mortality as a whole and in several cause groups of death in the first year of life in England and Wales. In the country generally within the 15 years studied there has been quite an appreciable decline at the rate of $3\frac{1}{2}$ per cent. annually in the total death-rate under one year. In the separate causes of death, however, there are great variations from this average rate of change. In view of the widely different sizes of the standard errors involved, it is impossible to say whether or not the actual magnitude of the coefficient of the regression line serves to place the separate cause groups of death in any definite order. Some causes have declined on the average much more than others, but the course has been very erratic. Good examples are furnished in the case of epidemic infectious diseases, e.g. measles, in which violent fluctuations may occur from year to year, although on the average of several years' experience a decline may be manifested. On the other hand with a group such as congenital debility the percentage decline may not appear so startling, but the consistency of the downward trend is a cause for satisfaction as great as, probably much greater than, a more decided decline but one subject to gross annual fluctuations from the general course. Probably all that can be done in this connection is to have three groups, namely those (1) with a significant decline, (2) with questionable or no decline and (3)/

(3) with an increase. In the first group the gastro-enteric infections, diarrhoea and abdominal tuberculosis, have declined most of all. Other causes included in (1) are overlying, meningitis, measles, atrophy, debility and marasmus, and infantile convulsions. Infectious diseases generally have been significantly reduced, but this is mainly attributable to the decline in measles mortality. Whooping cough, syphilis, bronchitis and pneumonia and premature birth, all of which show insignificant changes, compose group (2); whereas malformations and injury at birth have shown a definite tendency to increase.

(b) Ante-natal mortality. To complete the mortality list of infants it would be necessary to consider the death-rate in intra-uterine life. This, however, can only be done in a very imperfect way. Still-births, i.e. deaths after the 28th week of intra-uterine life, are notifiable, but since the introduction of notification there has, as will be gathered from Table 2, been no tendency for this rate to diminish.

TABLE 2.

Still-birth Rates per cent. of Live Births
in England and Wales.

1918	3.0	1922	3.2
1919	3.2	1923	3.3
1920	3.1	1924	3.3
1921	3.2	1925	3.2

It is only fair, however, to point out that the obvious conclusion from these figures is but doubtfully justifiable. Notification of still-births is not yet compulsory, and more complete returns might, with reason, be held to obscure a real downward tendency and to produce an apparently stationary or increasing/

increasing rate of mortality. No records are, unfortunately, available of the loss of foetal life before the attainment of viable age, and it is possible that this may be just as great as the more immediate ante-natal loss. A priori, it seems reasonable to suppose that the secondary sex-ratio (the sex-ratio at birth) would furnish a useful index of the total pre-natal loss of life. The sex-ratio at conception (the primary sex-ratio) is much higher than that at birth, and, since the intra-uterine mortality also is predominantly male, it might appear that the lower the secondary sex-ratio the higher had been the previous ante-natal loss of life. Parkes (1924) has shown that the foetal elimination in man is large (16.5 per cent. of all pregnancies), and quite sufficient, therefore, provided intra-uterine mortality is differential with regard to sex, to affect profoundly the sex-ratio at birth. That there is a definite excess of males among foetal deaths is borne out by all statistics. The male sex-ratio of live-births varies approximately between 104 and 108, whereas for still-births (Huxley, 1924) the male ratio lies between 124.6 and 142.2 in various European countries. The several factors which are known to influence the birth sex ratio, e.g. age and parity of the mother, social status, etc., do so probably more by varying the ante-natal loss of life than by modifying the sex proportions at conception. The experimental work of Parkes (1924, a) shows that, for mice at least, there is a strong degree of association between the secondary sex-ratio and the amount of foetal wastage. Adverse pre-natal environment accentuates the foetal loss and decreases the sex-ratio of live-births. Under normal conditions, in which the embryo loss amounts to 10.8 per cent., the sex-ratio is 118 males per 100 females/

females. In mice allowed to become pregnant immediately following parturition and to continue suckling the young for less than 6 days the embryo loss rises to 17.6 per cent. and the birth-ratio falls to 80.4. With more intensely adverse environmental conditions the foetal loss rises to 23.1 per cent. and the secondary sex-ratio falls to 62.1. If these results were applicable to man, the male ratio at birth would evidently provide a suitable index of ante-natal loss; but it is doubtful if the human secondary sex-ratio forms such a solid foundation for deduction as to the course of ante-natal mortality as would appear from results found under controlled laboratory conditions. The two following considerations give reason for this dubiety. In the first place the sex-ratio at birth of legitimate is almost equal to that of illegitimate infants. In the period 1911-25, of all legitimate births 51.10 per cent., and of illegitimate 51.06 per cent. were males. A higher pre-natal mortality is to be expected among illegitimates, because most of these mothers are primiparae and the dangers of first pregnancies are greatest; the ante-natal environment to which the infants are subject is probably much worse than is that of legitimate infants; and, still more, because a relatively great proportion is infected with venereal diseases. That the sex-ratios are so little different would rather seem to invalidate conclusions regarding the course of foetal loss which are drawn from the evidence of these ratios alone. In this instance, however, the lower average age of unmarried mothers will offset to an unknown extent the effect of those factors tending towards a low secondary sex-ratio among illegitimate infants. In the second place, there is no correlation between the notified still-birth rate and the secondary sex-ratio.

TABLE/

TABLE 3.

Correlations between the Still-birth Rates
and Secondary Sex-ratios (1921-23).

Metropolitan boroughs	.0496 ±	.127
County boroughs	-.0358 ±	.074
Administrative counties	.0840 ±	.102

Table 3 shows the coefficients of correlation* between these two variables found in the three large aggregates of England and Wales in the triennium 1921-23. These coefficients are neither significant with regard to their probable errors nor are they consistent with regard to sign, and therefore may be taken to indicate that the secondary sex-ratio is not related in any way to the preceding still-birth rate. Here again, however, we have really no strong argument against the sex-ratio at birth as an index of the total ante-natal loss, because we are ignorant both of the magnitude of the loss of non-viable foetuses and of the correlation between this and the death-rate in the period when viability has been attained. Taken together, however, these two findings cast reasonable doubt on the use of the suggested index of foetal wastage; but, for what it is worth, it may be said that, although there have been variations in the ratio of male: female live-births especially in the immediate post-war period, a regression line fitted to these ratios has a slope which is insignificant when compared with its standard error, i.e. there has been no significant general change in the sex-ratio at birth within this period. In so far, then, as this is indicative of what we want to measure, and taken in conjunction with the course followed/

* See Appendix.

TABLE 4.

Decline in Various Causes of Maternal Mortality
in England and Wales (1911-25).

Cause of death	Regression coefficient
Accidents of pregnancy	+ 0.82 ± 0.54
Abortion	+ 0.05 ± 0.90.
Ectopic pregnancy	+ 2.85 ± 0.61
Other accidents of pregnancy	+ 0.30 ± 0.96
Puerperal haemorrhage	- 0.59 ± 0.27
Other accidents of childbirth	+ 0.05 ± 0.48
Puerperal sepsis	+ 0.34 ± 0.62
Albuminuria and convulsions	- 0.63 ± 0.42
Phlegmasia alba dolens	- 1.01 ± 0.33
Puerperal insanity	- 1.87 ± 1.13
All causes	- 0.10 ± 0.27

followed by that part of the ante-natal death-rate which is notifiable, it may provisionally be concluded that no improvement has taken place at this period of infant life. The difficulties in the accurate measurement of the course of ante-natal mortality might make it seem unreasonable to draw any conclusions in this connection; but even if the evidence submitted only suggests, and cannot with certainty demonstrate a lack of improvement in the trend of foetal mortality, there are at least no certain grounds for the contrary opinion that there has been any amelioration.

(c) Maternal Mortality. The cognate subject of puerperal mortality must now be reviewed briefly. In Table 4 are collected the coefficients representing the course of the rates of mortality from all causes and from several separate cause groups of death in childbearing women. The coefficient for the total maternal death rate shows that its course in the period studied is practically horizontal, and that, therefore, there has been no change in the welfare of mothers as a whole. Among the separate causes of death, only two, namely phlegmasia alba dolens and puerperal haemorrhage, show sensible improvement; and even in these cases the coefficients are small and but just statistically significant. Causes of death in early pregnancy have shown a tendency to increase. In each instance the regression coefficient is positive in sign, although in only one case, that of ectopic pregnancy, can it be deemed substantial. Later in the child-bearing period the chief cause of death, albuminuria and convulsions, has shown an insignificant tendency to decline. As is well known, however, eclampsia (which forms the greater part of this group) is in the majority of cases a primiparous condition, and, since a declining birth-rate is associated with an increasing proportion of

of primiparous births, by basing the death-rate from this cause on total births, any tendency towards decline will be minimised and might be effaced. Thus if it were possible to calculate the mortality due to this cause on the more accurate denominator of primiparous instead of total births, it might quite well be that there has been real improvement. This possibility can only be mentioned because there are in this country no records of the number of primiparae. Apart from puerperal haemorrhage, causes of maternal death at and immediately after the birth of the child have remained practically steady within this period. The remaining, and chief single cause of maternal death, puerperal sepsis, has, if anything, shown a tendency towards increase, but no statistical significance can be attached to the coefficient. On the whole, therefore, we may conclude from a study of these figures that but little has been accomplished within recent years in promoting the well-being of the child-bearing woman.

To summarise briefly, it may be stated that, as regards maternity and child welfare in England and Wales generally, any significant improvement, so far at least as mortality is concerned, has been confined to the post-natal period of infancy. At and before the birth of the child conditions appear to have remained practically stationary in the last 15 years.

Discussion.

It remains now to seek some reasonable explanation of these changes. We have seen that the death-rates from various causes have declined at different rates; and we now wish to enquire how far these differences coincide with any reasonable classification of the causes of infant deaths. The simplest/

TABLE 5.

Tentative Classification of Principal Causes of
Infant Mortality (Pearl.)

A. Causes of death actually now well controlled or capable theoretically of direct control in greater or less degree.	B. Causes of death not controlled.
Measles	T.B. of lungs.
Scarlet fever	T.B. meningitis.
Whooping cough	Other forms of T.B.
Diphtheria and croup	Syphilis
Dysentery	Organic diseases of the heart.
Erysipelas	Malformations.
Tetanus	Premature birth.
Meningitis	Congenital debility.
Convulsions	Injuries at birth.
Acute bronchitis	
Pneumonia	
Bronchopneumonia	
Diseases of the stomach	
Diarrhoea and enteritis	
External causes	

simplest would be a dichotomy of the causes into avoidable and unavoidable; and, if it could be shown that the decline in infant mortality had been confined to the avoidable group and left unaffected the non-preventible group, there would then be some basis for the belief that the decline had been due in greater part to intentional efforts of control. Although in the majority of cases no difficulty will be found as to which of these two classes any cause of death should be relegated, there will always be a residuum which leaves room for ^{w/h} difference of opinion. Take, for example, the following tentative classification (Table 5) given by Pearl (1920). We may reasonably object to this classification on the following grounds. (a) In the group of causes of death not controlled, the inclusion of tuberculosis probably does not accord with the greater part of medical opinion. The main type of infection at this period of life is either abdominal or meningeal and not pulmonary, and although the effects of treatment of adult tuberculosis are doubtful, efforts to prevent infant tuberculosis, which is caused rather by ⁷ ingestion than inhalation of tubercular material, should be more successful. Control of the milk supply and proper infant feeding should surely do something to lessen this cause of mortality. (b) Also in this group is included infant syphilis, with regard to which the results of treatment are admirable. For example, Sir George Newman (1926) quotes some figures relating to the treatment between 1917 and 1924 of 164 women, most of whom were suffering from secondary syphilis. There were 152 babies born, 51 with positive and 101 with negative Wasserman reactions. Of these infants 12 were stillborn as treatment was commenced too late in pregnancy, 4 babies died within/

fallen is no certain reason for the belief that it is controlled or capable of control; and to arrogate to intentional efforts, medical or other, any such amelioration without clear statistical proof of the efficacy of the remedy gives rise to hopes which are only dispelled by a recrudescence of the disease in spite of the continued application of orthodox methods. The history of scarlet fever, the mortality from which has declined so greatly and the reasons for this still puzzle epidemiologists, is a striking example of the foregoing objections, and in itself sufficient to make us cautious of accepting Pearl's classification.

But although there are many difficulties in the way of classifying causes of death in infancy, it is certainly a necessary procedure before we can offer any satisfactory explanation of the factors which have been responsible for the changes in the mortality rates of infancy. The difficulty is to put forward any other classification for which we can offer statistical justification. In assessing the values of maternity and child welfare schemes, for example, there are, apart from prosecuting local, carefully controlled ad hoc inquiries, only two methods of inquiry available, both of which are inferior to local investigation. One of these depends, as above, on arranging the causes of mortality in the order in which they might be expected to be affected - a matter of personal opinion; the other is to compare the rates of decline in parts of the country in which the extent to which these schemes have been prosecuted differs. So far as those causes of death which might reasonably be expected to react favourably to ante-natal supervision, namely ante-natal and natal deaths of infants and the mortality of childbearing women, there is no evidence that any appreciable amelioration has taken place (Tables 1 - 3). The second method, that of interlocal comparison of the rates of/

TABLE 7.

Decline in Various Causes of Infant and Maternal Mortality in
Different Parts of England and Wales. (1911-25)

Cause of death	London	County boroughs	Urban districts	Rural districts
Infant mortality	-3.95±0.63	-5.42±0.44	-5.87±0.54	-3.04±0.38
Tuberculosis (all forms)	-8.40±1.10	-7.79±0.78	-7.81±0.79	-7.28±0.68
Diarrhoea	-8.43±2.14	-9.05±2.51	-10.53±5.26	-9.28±2.75
Infectious diseases	-5.73±1.95	-4.16±1.34	-5.18±1.62	-5.24±1.28
Measles	-6.61±2.74	-6.25±1.65	-7.79±2.44	-8.06±2.47
Whooping cough	-1.89±2.37	-2.78±1.72	-3.73±1.62	-4.19±1.32
Bronchitis and pneumonia	-1.85±0.99	-0.66±0.56	-1.58±0.70	-2.15±0.78
Abdominal tuberculosis	-13.84±1.77	-11.90±1.13	-11.16±1.31	-10.94±1.10
Syphilis	-5.65±1.55	-2.25±1.18	-2.71±1.21	+0.78±1.50
Convulsions	-8.34±0.56	-7.21±0.28	-6.03±0.57	-5.99±0.39
Malformations	+0.05±0.51	+1.25±0.30	+0.95±0.31	+1.15±0.36
Injury at birth	-0.23±0.73	+2.77±0.61	+2.79±0.58	+1.85±0.55
Premature birth	-1.45±0.42	-0.63±0.23	-0.64±0.31	-0.33±0.22
Atrophy, debility and marasmus	-6.51±0.70	-6.63±0.57	-6.44±0.57	-5.59±0.42
Meningitis	-8.13±1.83	-7.42±0.62	-7.47±0.60	-5.35±0.90
Overlying Maternal mortal- ity	-11.61±1.04	-9.75±0.81	-5.68±0.63	-3.46±0.73
Accidents of pregnancy	+0.10±0.24	+0.23±0.37	-1.12±0.65	-0.48±0.33
Puerperal haemorrhage	-0.54±1.17	+1.41±0.71	+1.05±0.51	-0.36±0.66
Other accidents of childbirth	+1.89±0.91	-1.04±0.57	-0.54±0.53	-1.49±0.81
Puerperal sepsis	+0.68±1.28	-0.28±0.51	-0.31±0.61	+0.94±0.71
Albuminuria and convulsions	-0.31±0.77	+1.14±0.68	-0.35±0.64	+0.28±0.71
Phlegmasia alba dolens	+0.56±1.18	-1.15±0.70	-0.82±0.49	+0.01±0.46
Puerperal insanity	+0.51±1.16	-0.52±0.44	-1.38±0.65	-1.63±0.68
	-3.31±3.04	-0.16±1.54	-1.62±2.18	-4.56±2.61

of decline, is also not without difficulty. There are no exact means of measuring the extent to which preventive work has been carried on in different districts; so that the comparisons must necessarily be crude. The number of ante-natal or child welfare centres in the large aggregates of the country is an imperfect measure of the amount of work done because of the differences in the populations to be supplied. The number of clinics per 1000 births or the average number of visits to a clinic per birth are better, but still doubtful indices, since they take no account of the work done by health visitors. The average number of ante-natal and infant welfare clinics per 1000 births in the three large aggregates of England and Wales are given on Table 6.

TABLE 6.

Number of Ante-natal and Infant Welfare Centres per 1000 Births. (Average 1924- 6.)

	Ante-natal	Infant welfare
London	1.40	2.70
County boroughs	0.75	1.88
Administrative counties	1.03	3.69

To compare with these indices, the rates of decline for the separate causes of death in maternity and infancy are given on Table 7. The feature of interest here is that both as a whole and in all the separate cause groups of death examined there are few instances of a statistically significant difference in the rates of decline in the main subdivisions of the country. The differences which are evident, however, though small, are of importance. Syphilis and premature birth show substantially more/

more decline in London than elsewhere, and injury at birth in London, although for all practical purposes stationary, compares favourably with the significantly positive coefficients in the rest of the country. The death-rate from congenital malformations, too, which has remained unchanged in London, shows significant increase in other subdivisions of the country. In two other causes of infant mortality, convulsions and overlying, local differences in the rates of decline are apparent. In both instances the coefficient of regression is lowest in rural districts, somewhat higher in urban districts and highest of all in County Boroughs and London. Although the differences in the rates of decline in the death rates from syphilis, injury at birth and premature birth would appear to be related to the amount of ante-natal work done (as measured by the imperfect index given above), it is evident from the size of the coefficients how little the total mortality from these causes has been affected. Further, since there are no indications of any similar relationship in the cause groups of maternal mortality between the changes in the death rates and the extent of ante-natal supervision, it is possible that other factors are at the root of these changes. For example, the differences found in such causes as syphilis, premature birth and possibly congenital malformations might be due to the lessened incidence or more effective treatment of venereal diseases in the adult population. The results contained in Tables 6 and 7, therefore, are at the best only doubtful evidence of correspondence between effort and result.

In suggesting any other classification of causes of death, it should be remembered that there are three main external factors on which the life of the infant depends:-

(1)/

- (1) The care bestowed on it by the mother.
- (2) Medical care, or the neglect to call in medical assistance in illness - a factor which is obviously related to the intelligence and efficiency of the mother, and
- (3) Environment other than maternal.

It is not pretended that this is the order of importance of each of these, or that these are the only influential factors; but since these are certainly among the most important, and since it is possible to separate each of these in some degree by comparison of figures from the Annual Reports of the Registrar-General, it seemed of interest to study them in relation to the changes in mortality already referred to.

The effect produced by a combination of maternal neglect and lack of proper medical care may be studied best on a large scale by a comparison of the rates of mortality from separate causes of death among legitimate and illegitimate infants. The illegitimate infant is unwanted, and so long as it lives there attaches to the mother a stigma which only the death of the infant can efface. The baby, therefore, in many cases is deprived of the benefits of both intelligent maternal and medical attention.

The special features connected with the evils of unfavourable surroundings can be demonstrated by comparing the death-rates by causes in the County Boroughs of the North with those of the Rural Districts of the South of England. It is not to be expected that these two comparisons will show any clear-cut division, because maternal neglect and unhealthy environment are to some extent correlated; but by studying the differences between these two, and comparing them with the observed rates of decline in the various causes of infant mortality, we may be enabled to reach some fairly definite conclusion.

These/

TABLE 8.

Indices of (1) Adverse Environmental Conditions and
(2) Maternal Neglect.

(1) = Ratio per cent of Death-rates of County Boroughs of
the North: Rural Districts of the South.

(2) = Ratio per cent of Death-rates of Illegitimate: Legitimate
Infants.

	(1)		(2)
Measles	955	Syphilis	732
Diarrhoea and enteritis	365	Overlying	316
Syphilis	359	Atrophy, debility and marasmus	253
Bronchitis and pneumonia	317	Diarrhoea	240
Infectious diseases	268	Abdominal tuberculosis	228
Infantile convulsions	202	Infantile convulsions	179
Tuberculosis (all forms)	193	Measles	175
Meningitis	186	Tuberculosis (all forms)	167
Whooping cough	175	Premature birth	166
Atrophy, debility and marasmus	144	Injury at birth	162
Premature birth	139	Bronchitis and pneumonia	144
Overlying	112	Infectious diseases	142
Malformations	110	Whooping cough	124
Injury at birth	110	Meningitis	116
		Malformations	115

These two comparisons are given on Table 8. As might have been expected from the nature of the case, syphilis heads the list in column (2). The ratio found may be far higher than is actually the case, both because of the unwillingness to certify this as a cause of death (more especially when the birth is legitimate), and because a greater proportion of illegitimate births occur in hospital where diagnosis and certification are usually more accurate; but in all probability syphilis is the cause which has the greatest excess among illegitimate babies. Among other causes it will be seen that some of the groups occupy high positions in both columns, but that the chief differences are (a) overlying and atrophy, debility and marasmus, both of which groups occupy high places in column (2) and low places in column (1); and (b) infectious diseases as a whole, measles, bronchitis and pneumonia and whooping cough, all of which have noticeably higher places in column (1) than in column (2). Unfortunately abdominal tuberculosis is not given separately in the Registrar-General's reports for County Boroughs of the North and Rural Districts of the South, so that this cause must be omitted from consideration.

If the chief causes of the decline in infant mortality had been those measures designed to lessen or remove the evils of insanitary and overcrowded surroundings, then we should expect that those diseases in which the proportional excess was greatest under these conditions would have declined to a greater extent; but as will be seen on reference to the table of regressions (Table 1) numerous exceptions to this rule occur. In particular, bronchitis and pneumonia, infectious diseases generally, atrophy, debility and marasmus and overlying may be mentioned. Comparing now the observed decline in the death/

death-rates with the position of the diseases occupied in column (2), Table 8, we find a much more striking agreement. Here we have only two exceptions, namely meningitis not of tubercular origin, and syphilis. It is not pretended that the decline is in exact relation to the position occupied by the disease in column (2). Some of these cause groups of death will be observed to have declined on the average to the same extent, but the wide differences in the standard errors, which are a measure of the stability of the decline, show that the same significance cannot be attached to those in which the slope of the line is the same. Why meningitis should upset the relation we have no means of discovering, but the disease is not numerically an important cause of mortality, and is one of those cause groups which may be influenced by difficulties in diagnosis especially with infectious diseases in childhood. With syphilis, of course, we are on entirely different grounds. That the death-rate from this condition has not declined is not at all the fault of the mother. Even the most intelligent and careful woman will not be able to do anything towards curing herself or her child of the disease without the aid of efficient therapeutic remedies, and that this death-rate has not come down only argues that direct medical treatment is not even one of the important causes of the recent decline in the mortalities of infancy.

The previous discussion, however, suggests at least one method by which a rational classification of the causes of infant death may be made. The classes which the comparisons indicate are three, namely:-

(1) Capable of Direct Control. Those conditions in which medical and surgical measures can effect a cure or sufficient amelioration to prolong the life of the individual and in which maternal care/

care of itself is of no avail. In this group we would include infantile syphilis, injuries at birth, diphtheria, and some causes of still-birth and premature birth, such as eclampsia and antepartum haemorrhage.

(2) Capable of Indirect Control (and chiefly by proper maternal care). Here we would include tuberculosis in all its forms in infancy but more especially the abdominal type, diarrhoea and enteritis, atrophy, debility and marasmus, overlying and probably convulsions.

(5) Uncontrollable Causes. Infectious diseases, such as whooping cough, scarlet fever etc., malformations, meningitis and premature birth from causes not included in (1), probably also measles and bronchitis and pneumonia. The fairly high position in column (2) of premature birth might make it doubtful if this should be included here; but it is probable that some of this excess in itself represents indirectly the greater prevalence of venereal infection among illegitimate babies, miscarriage and premature birth being common features in the obstetric history of a syphilitic mother.

Although objections may well be raised in some respects to this classification, it at least possesses some statistical basis, and moreover, it must be recognised that it is difficult, if not impossible, to draw hard and fast lines between the groups. All that is suggested is that in controllable illness in infancy and childhood, there is to be considered the effect which a good mother may have in preventing the disease, and also that there are certain conditions in which, no matter how intelligent and careful the mother be, the end result can only be influenced by skilled medical treatment; and the classification given here simply suggests which, in my opinion, of these two factors is the more important in the several causes of/

of death. It may be noted, however, that the classification states implicitly that the evil effects of domestic and municipal overcrowding and insanitation can, except in the case of infectious disease, be overcome by intelligent and conscientious motherhood.

If such a classification be permissible and is anywhere near the truth, then within the last 15 years, group (2) is the only one which has shown any significant decline, whereas groups (1) and (3) have remained practically stationary.

Sir George Newman (1920), in reference to the health of the mother and child remarks (p.44); "Immense progress has been made in this subject within recent years. The Midwives Act of 1902, the School Medical Service organised in 1907, the Insurance Act Provisions for Maternity Benefit in 1911, and the Maternity and Child Welfare Act of 1918 are the mile-stones of a forward-looking nation. The results have been very remarkable. The decline in the death rate of infants from 150 per 1000 born in 1900 to 89 in 1918 must be attributed in large measure to the action taken. It is one of the two or three significant triumphs of preventive medicine in the present century." Further, he says; "It is a movement springing in a large degree from the people themselves, and resulting in a new social conscience in respect of the physical well-being of mothers and children."

The two statements are mutually contradictory unless they are related as cause and effect. There is no doubt about the amount of administrative action that has been taken. The difficulty is to know if it has had any effect. It has been taken too much for granted, I think, that welfare work and the decline in infant mortality are causally-related phenomena. The decline which has taken place in the different causes of death among infants serves to show clearly that in diseases amenable to medical/

medical treatment alone (group 1 of the suggested classification) practically nothing has been accomplished, and that the causes which have declined are those in which the main factor responsible is probably the mother herself. It is, moreover, worthy of note that the decline which has taken place in the death-rates of infancy began before the introduction of deliberate national efforts at teaching or treating mothers, and that inspection of the death curve shows no acceleration in the rate of decline on the introduction of these measures. I am well aware of the fallacies inherent in this type of argument and that legislative enactment in many instances only represents the climax of a steadily increasing amount of private philanthropic work; but this point should be remembered and carry some weight in any discussion of the subject. Further, although more adequate maternal care bestowed on the infant may in all probability be the result of welfare work - the birth and growth of a new social conscience among present-day mothers - we must not overlook the fact that a similar result may follow on deliberate restriction of the size of the family to the desired number. The precise interpretation of the relation between the birth and infant mortality rates is not a simple one, but it does seem probable that limitation of the number of children born may in part be due to a praiseworthy attempt of the parents to provide the family with the best prospect of survival and success in life. If, however, the decline in the mortality of infancy can be justly attributed to deliberate attempts at preservation of infant life, it follows that the link between supervision and facilities for adequate treatment is missing, and that with more effective coordination we may hope for a still further amelioration.

III. THE RELATIVE IMPORTANCE OF MATERNAL HEALTH, SKILLED OBSTETRICAL ASSISTANCE AND EXTERNAL ENVIRONMENTAL CONDITIONS IN THE CAUSATION OF THE GEOGRAPHICAL VARIATIONS OF THE STILLBIRTH, NEO-NATAL, AND POST-NATAL DEATH RATES.

Although within recent years the risk to infants has diminished considerably, the rate of mortality in the first year of life is still higher than in any other period of life prior to that at which we can hope for little more reduction. That there is scope for further improvement in the mortality experienced in early life is unquestioned, therefore, a brief review of the statistical data relating to England and Wales and a study of some of the factors of causal import may not be inappropriate.

Since the beginning of the present century infant mortality has shown a considerable decrease in all areas of the country. Previously, the death-rates at later ages had already been falling, but the fate of infants and young children had failed to follow a similar course. In 1901, a fairly abrupt change occurred, and since then infant mortality has steadily declined, the rate of decline having shown no tendency to decrease, apart from minor annual fluctuations, until the last few years.

If the death-rate under one year be further subdivided, it is found that the mortality experienced in the first few days of extra-uterine existence is far greater than at any other period, and from that point rapidly declines, the death-rate in the last three months of the first year of life being only about 3 per cent. of that experienced in the first week, when reckoned per unit of time. A closer inspection of the trend of mortality at various ages under one year shows that all ages have not shared in this amelioration to the same extent/

extent. Each age period has been affected in varying degrees. The data presented in Table 9 show that, as a general rule, the nearer to birth the less has the mortality rate been affected.

TABLE 9. The distribution in age periods of the infant mortality rates per 1000 births.

	Under 1 month	1-3 months	0-3 months	3-6 months	6-12 months	Under 1 year
1901-05	-	-	70	28	40	138
1906-10	40	23	65	22	32	117
1911-15	39	20	59	20	51	110
1916-20	37	17	54	14	22	90
1921-25	53	13	46	12	18	76

Under one month the rate of mortality between the quinquennia 1906-10 and 1921-5 declined $17\frac{1}{2}$ per cent, at one to three months $43\frac{1}{2}$ per cent, all under three months 36 per cent, three to six months 57 per cent, and six to twelve months 55 per cent. The time of the appearance of this general change, however, was approximately the same in each of the separate age groups under one year, the death rates having risen to a maximum in the quinquennium 1896-1900. As a consequence of this differential decline, the proportion of deaths which occur at those ages has changed significantly, the deaths at, and shortly after birth now forming a more important contribution to the total death roll.

These differences in the behaviour of the death-rates at various ages under one year suggest that at each of these ages, the several factors in the causation of infant mortality differ in their importance at each of these age groups. This we already know from general medical knowledge to be probably true. In early infancy, the effects of pre-natal influences will scarcely have worn off, but the more distant from birth, the less important/

important are such factors likely to become. The effects of variations in obstetrical assistance (using the term in its widest sense) are also likely to be most clearly reflected on the mortality at and immediately succeeding birth, whereas maternal and environmental influences would appear to become of more and more importance as infancy advances.

It seems fairly obvious, therefore, that the death-rate of infants under one year cannot be taken as a compact group and satisfactorily investigated as such. In infant life, at least four broad stages, perfectly definite in character although overlapping one another in time to a greater or less degree, can be recognised. First of all is the foetal or parasitic stage, extending from the period of conception until immediately the infant is born. Secondly, and included in the first, are the several stages of labour itself. Thirdly, a period of adjustment to a new mode of life which consists of the first few days or weeks of independent extra-uterine existence, and lastly, there is the remainder of infant life. There can be no doubt that each of these periods represents entirely different phenomena, and the mortality at each of these stages seems to require special investigation.

From the statistical returns of the Registrar-General and the Annual Reports of the Chief Medical Officer of the Ministry of Health, it is fairly easy to obtain a subdivision of the mortality of infants into parts which at least approach the classification suggested. No account can be taken of the loss of possible lives in early foetal life (abortions, etc), but the notifications of still-births published in appendices to the Annual Reports of the Chief Medical Officer of the Ministry of Health represent, as accurately/

accurately as can be done at the present time, the deaths of foetuses at any period between the 28th week of intra-uterine life and full time. Although in some parts of the country notifications may be somewhat defective, since these are not yet compulsory, the figures given probably represent fairly accurately the distribution of still-births throughout the country. The death rate of infants born alive may be subdivided simply into two categories, (a) the death-rate from "congenital debility, malformation and premature birth" (Number 28 of the short list of causes of death given for each separate district in the Annual Reports of the Registrar-General), and (b) the remainder of infant deaths under one year. Group (a) consists of deaths from accidents of birth, prematurity, atelectasis, hydrocephalus and other developmental defects. This, for convenience, will be referred to as the neo-natal death rate. The remainder of infant deaths are mainly the result of gastro-enteric, respiratory and infectious diseases. These will be referred to as the post-natal death rate. Although no separate account can be taken of the deaths during the processes of labour itself, it will be seen that this subdivision into three groups corresponds fairly well with the stages of life suggested. Besides representing to some extent a biological classification, it also represents fairly well a temporal subdivision, since the greater part of the area of the death curve from neo-natal causes is contained in the first month of life, whereas the common infectious, respiratory and enteric diseases are not very prevalent at this time.

Of the numerous possible factors which may affect infant life in each of these stages, at the present time available/

available national statistics only permit of an attempt to measure three of the presumably chief ones:-

- (1) The quality of the obstetrical assistance in childbed.
- (2) The health of the mother, and
- (3) Social and environmental conditions.

As a rough measure of (1) the total maternal mortality rate in childbearing has been taken; but, since a great part of this death-rate is formed by the group of conditions under the term puerperal sepsis, and since this is not determined to any significant extent by the proximity to medical aid in childbearing, the two subgroups of the mortality in childbearing, puerperal sepsis and the remainder, have, in addition, been considered separately. As an index of the general level of the health of mothers in any district, the death-rate in women aged 15-45 from all causes less those connected with childbearing has been used as the most exact measure which is available in any statistical returns. For social and environmental indices, the proportion of female indoor domestic servants per 1000 of the total population, the proportion of the population living more than two in a room, and the number of rooms per person have been used as general measures of these factors. Still-birth returns are not available for urban and rural districts separately, so that these have only been investigated in the counties of England and Wales, and separately for county boroughs. The remaining two groups of infant deaths have been analysed in counties, county boroughs, urban and rural districts. The rates of mortality have been calculated on the births and deaths occurring in the triennium 1921-3.

TABLE/

TABLE 10. Means, standard deviations and coefficients of variation of the three infant death-rates.

	Mean	Standard deviation	Coefficient of variation
Ante-natal Mortality	31.08	6.65	21.41
Neo-natal Mortality	30.54	3.50	11.45
Post-natal Mortality	38.08	10.92	28.67

(a) Counties. The results for counties will be considered first. In Table 10 are given the mean death-rates, their standard deviations and coefficients of variation. Calculated per 1000 births, the post-natal death-rate of infants is highest, and, both absolutely and relative to its mean value, the most variable. The average ante-natal death-rate is much lower than this, and is also less variable. The neo-natal death-rate is lowest of all, not only in its mean value, but also in its dispersion throughout the country. This comparison of averages, however, obviously gives a wrong impression of the force of mortality at these periods. The still-birth rate is calculated on all deaths which occur in the three months before full time, as well as those occurring during labour but before the child is born; the neo-natal death-rate is (roughly) confined to the first month after birth, and the remainder of the infant death-rate is spread over the remaining eleven months of the year; so that if these separate mortality rates be reckoned approximately as death-rates per 1000 births per annum, the mortality experienced in the short period succeeding birth is 360 per 1000 births, the ante-natal mortality 124 per 1000, and 40 per 1000 per annum in the remainder/

remainder of infant life. (These figures are only crude approximations to the truth, as they are based on the false assumptions that the frequency distributions of these deaths at the three periods of life are rectangular and do not overlap one another; but they are sufficient to demonstrate that the force of mortality, the death-rate per unit of time, in the period immediately subsequent to birth is about three times as great as that in the three months preceding birth and about nine times as high as that which occurs later in the first year of life.)

The crude correlation coefficients between these three death-rates and the factors being considered in relation to them are given in Table 11.

TABLE 11. Coefficients of correlation between each of the infant death rates and certain influential factors.

	1. Ante-natal mortality	2. Neo-natal mortality	3. Post-natal mortality
4. Death-rate in females, 15-45	.391 \pm .087	.624 \pm .063	.817 \pm .034
5. Maternal mortality rate	.508 \pm .076	.429 \pm .084	.492 \pm .078
6. Puerperal sepsis death-rate	-.055 \pm .103	.133 \pm .101	.488 \pm .078
7. Maternal death-rate less sepsis.	.603 \pm .065	.418 \pm .085	.510 \pm .095
8. Per cent. living more than two per room	-.004 \pm .103	.440 \pm .085	.655 \pm .059
9. Domestic servants per 1000 population.	-.224 \pm .098	-.654 \pm .059	-.724 \pm .049

From these it will be noted that the health of mothers is most closely related to the post-natal death-rate, less so with the neo-natal death-rate and least of all with the still-birth rate. With each of the death-rates, the correlations are certainly significant with regard to the probable errors involved.

The/

TABLE 12. Coefficients of correlation.

Variables	Correlation
Female death rate, 15-45, and maternal mortality rate	.4202±.085
" " " " puerperal sepsis death rate	.3717±.089
" " " " maternal mortality less puerperal sepsis	.3045±.093
" " " " female indoor domestic servants	-.7320±.048
Female indoor domestic servants and maternal mortality rate	-.2264±.098
" " " " puerperal sepsis death rate	-.3701±.089
" " " " maternal mortality less puerperal sepsis	-.0857±.102

The total mortality rate of women in childbearing, curiously enough, shows but little difference in its relationship with the three groups of infantile deaths, a result which, if the maternal mortality rates were an exact index of the state of obstetrical supervision, would immediately lead to the suspicion that an indirect association due to some other factor had produced the result. But if the mortality due to puerperal sepsis be excluded, the remainder of the death-rates of women in childbearing becomes less associated with the death rates of infants as age advances. In all instances the coefficients are significant, but the relationship with still-births is highest and that with the post-natal death rate lowest. Comparing the coefficients with those for health of the mother, the coefficients involving maternal health are, with still-births, lower than for those with the index used for the availability of obstetrical assistance; but, in the neo-natal and post-natal death rates, maternal health shows higher correlations than does the death rate of women in childbearing.

With external environmental and social conditions there is no significant relationship with the ante-natal death -rate, but fairly high and certainly significant relations with both the neo-natal and post-natal mortalities, the post-natal rate showing a slightly closer association with both indices of environment than does the neo-natal death rate.

All the variables we are studying in relation to the mortalities of infancy are, as will be gathered from the coefficients collected in Table 12, interrelated to some extent, environment and the general health of women fairly closely/

closely, maternal mortality significantly with maternal health and only slightly with environment, whereas the real criterion of obstetrical facilities (i.e. deaths in childbearing less puerperal sepsis) is less correlated with maternal health than is the total maternal mortality rate, and not at all with environment. Accordingly it seems of interest and importance to ascertain the extent to which each of these factors is related to the infant death rates when due allowance has been made for any indirect association which may be introduced by the correlation among the variables themselves; i.e. to calculate the coefficients of partial correlation* between each of the infant death rates and any one of these factors for constant values of the remaining two. In deducing the partial correlations, the number of domestic servants per 1000 population has been taken as the measure of environment. The results are collected in Table 13.

TABLE 13. Coefficient of partial correlation.

$r_{14.59} = .195 \pm .099$	$r_{24.59} = .159 \pm .100$	$r_{34.59} = .524 \pm .075$
$r_{15.49} = .404 \pm .086$	$r_{25.49} = .309 \pm .093$	$r_{35.49} = .348 \pm .090$
$r_{19.45} = .050 \pm .103$	$r_{29.45} = -.417 \pm .085$	$r_{39.45} = -.578 \pm .088$

The subscripts are:

- | | |
|--------------------------|--|
| 1 = Ante-natal mortality | 4 = Death rate females, 15-45. |
| 2 = Neo-natal " | 5 = Maternal mortality rate |
| 3 = Post-natal " | 6 = Domestic servants per 1000 population. |

These coefficients suffice to show that, of those investigated here, the important factor in determining still-birth mortality is the quality of obstetrical aid in child-birth. Maternal health shows a slight but insignificant positive relationship, and environment none at all.

The neo-natal death rate shows about equal relationship with environment and the maternal mortality rates in childbearing/

* See Appendix

childbearing; but the health of the mother is not significantly related to the mortality rate at this period of infant life.

The post-natal death rate is most closely connected with variations in the health of women and somewhat less so with both environment and the total maternal mortality rate in childbed. It will also be noted that the partial correlation between the post-natal death rate and the childbearing mortality rate for constant values of environment and maternal health is of the same order of magnitude as that found for both the neo-natal and the still-birth rate. That there should be such a high residual correlation with the mortality of women in childbearing and the infant death rate at this stage of life is certainly contrary to what would have been predicted. We should expect that the effects on the mortality of infants of the quality of the assistance afforded to women while pregnant should diminish as the age of the infant increases; but from the results it would appear that this is not so. There are two possibilities, however, which must be borne in mind in this connection. (1) In places where many women lose their lives in childbed, many more are rendered invalids for a time from the effects of causes which they managed to survive, but which killed others, and because of this many infants must be deprived of proper maternal care. (2) The total death rate of women in pregnancy and parturition is being used here as a measure of the quality of the assistance provided for the mother during pregnancy and in labour; but, as has already been pointed out, it is only a rough measure of this factor, and we must not overlook the fact that it may equally well serve as a measure of some other factor which is associated not only with deaths of women in childbed, but also with some causes of infant deaths. For instance in the present/

present case, it might be suggested that some infant deaths late in the first year of life may be dependent on the ease with which competent medical assistance can be obtained. Cases such as these can easily be adduced. Here, then, it is quite possible that the correlation may simply demonstrate a relationship between deaths which are preventable by timely medical intervention. But, before considering either of these possibilities, it seems advisable to enquire if similar results would be produced by taking the mortality rates in childbed from causes other than puerperal sepsis as indicative of variations in the available obstetrical facilities. Consequently the partial correlations have been recalculated using this variable instead of the total maternal mortality rate, and these are given in Table 14.

TABLE 14. Coefficients of partial correlation .

$r_{14.79} = .172_{\pm.099}$	$r_{24.79} = .135_{\pm.101}$	$r_{34.79} = .552_{\pm.071}$
$r_{17.49} = .546_{\pm.072}$	$r_{27.49} = .424_{\pm.084}$	$r_{37.49} = .194_{\pm.099}$
$r_{19.47} = -.021_{\pm.103}$	$r_{29.47} = -.468_{\pm.080}$	$r_{39.47} = -.356_{\pm.090}$

The subscripts are:

- | | |
|--------------------------|---|
| 1 = Ante-natal mortality | 4 = Death rate females, 15-45 |
| 2 = Neo-natal " | 7 = Death rate from causes in childbearing less puerperal sepsis. |
| 3 = Post-natal " | 9 = Domestic servants per 1000 population. |

From these it will be seen that the coefficients involving this portion of the maternal death rate are now higher in both the ante-natal and neo-natal mortalities, but with the post-natal mortality the correlation has now no statistical significance. It may be concluded, then, that the provision of assistance to pregnant and parturient women is in counties/

counties reflected on the still-birth rate and slightly less so on the neo-natal mortality rate, and that after approximately the first month of life, the effects of this factor cease to have any significant influence on the death rates of infants.

Among infants born alive, it is to be noted that environment plays a greater part early in life than does the health of the mother, which at this period of infancy is apparently of no great importance, whereas after the neo-natal stage of life has been passed, the health of the mother would appear to take slight precedence over external environmental influences.

Finally it remains to be determined what is the sum total effect of these three variables on the death rates at each of these three periods of infant life. Adopting Yule's (1922) notation, the multiple correlation coefficients* between each of the infant death rates and these three factors have been calculated and are given in Table 15.

TABLE 15. Coefficients of multiple correlation.

R	1.459	=	.403±.086
R	2.459	=	.556±.071
R	3.459	=	.698±.053

The coefficient is smallest for the still-birth rate, slightly higher for the neo-natal death rate and greatest for the post-natal death rate. A similar result is obtained if, instead of using the total maternal mortality rate, we use the death rates from all childbed causes less puerperal sepsis. These are given in Table 16.

TABLE 16. Coefficients of multiple correlation.

R	1.479	=	.483±.079
R	2.479	=	.585±.068
R	3.479	=	.681±.055

An insignificant increase results in the total coefficients/

* See Appendix

coefficients for the still-birth and neo-natal mortality, and an equally insignificant decrease with the post-natal death rate .

Assuming that variations in each of these three variables are capable of being to a great extent eliminated, in the case of environment by suitable administrative measures and in the remaining two, the health of the mother and the quality of assistance provided for the mother at the birth of the child, by organised medical efforts of control, these results show that least of all can be hoped for by improvement of these factors in the case of still-births, somewhat more with neo-natal deaths and most of all with the post-natal death rate. Although this is by no means a complete catalogue of all the conditions influencing infant life, and although the variables used here are, at their best, but imperfect measures of the factors we wish to investigate, these results fully demonstrate that there is still a problem to be solved. By eliminating fluctuations in all of these three important factors, the variations in the mortalities of infancy can only be reduced in the case of still-births by 12 per cent, neo-natal deaths 19 per cent and post-natal deaths 27 per cent. Even if this could, therefore, be effected there would still remain appreciable differences, presumably determined by measurable causes, in the rates of mortality in different parts of the country.

(b) County boroughs. Since data similar in all respects to those already examined for counties are available for the separate county boroughs of England and Wales, the whole series of constants has been recalculated for these districts in 1921-3 for purposes of comparison.

The/

The mean death rates at each of the three periods of infant life and the variability in the distributions throughout the country are given in Table 17.

TABLE 17. Means, standard deviations and coefficients of variation of the three infant death rates in county boroughs.

	Mean	Standard deviation	Coefficient of variation
Ante-natal mortality	35.18	9.75	27.70
Neo-natal mortality	33.27	5.55	16.62
Post-natal mortality	49.52	13.19	26.63

Comparing these figures with those for counties, it will be noted that all the death rates are higher in large towns than in the country generally, but that the difference is greatest in the case of the post-natal death rate. In county boroughs the death rate at this period is 30 per cent. higher than in counties, whereas the still-birth rate is only 13 per cent. and the neo-natal mortality only 9 per cent. higher than in counties.

Evidently, then, the conditions in urban communities prejudicial to infant life are reflected most clearly on the post-natal death rate, less so on the ante-natal, and least of all on the neo-natal death rate. With regard to variability in the rates of mortality, in county boroughs the coefficients of variation of the ante-natal and post-natal death rates are equal in magnitude, whereas in counties the post-natal death rate showed a slightly greater dispersion than the still-birth rate. The neo-natal death rate, both absolutely and relative to its mean value, in county boroughs is only slightly more variable than in counties.

TABLE/

Table 18. Coefficients of correlations between each of the infant death rates and certain influential factors in county boroughs.

	Ante-natal mortality	Neo-natal mortality	Post-natal mortality
Death rate in females, 15-45	.241 \pm .070	.418 \pm .061	.707 \pm .057
Maternal mortality rate	.383 \pm .064	.539 \pm .066	.183 \pm .072
Puerperal sepsis death rate	.280 \pm .069	.189 \pm .072	.153 \pm .075
Maternal death rate less sepsis	.319 \pm .067	.244 \pm .070	.109 \pm .074
Rooms per person	-.114 \pm .074	-.612 \pm .047	-.971 \pm .005
Domestic servants per 1000 population	-.171 \pm .072	-.437 \pm .060	-.658 \pm .042

The crude coefficients of correlation between the three divisions of the infant death rate and the variables already referred to are given in Table 18. These results compare very well with those already found for counties, and, although in most instances the coefficients seem smaller, none of the observed differences is of any statistical significance.

TABLE 19. Coefficients of partial correlations in county boroughs^x.

$r_{14.59} = .156\pm.073$	$r_{24.59} = .173\pm.072$	$r_{34.59} = .491\pm.057$
$r_{15.49} = .359\pm.065$	$r_{25.49} = .312\pm.067$	$r_{35.49} = .106\pm.074$
$r_{19.45} = -.050\pm.074$	$r_{29.45} = -.261\pm.069$	$r_{39.45} = -.395\pm.065$

^xThe subscripts have the same meaning as the counties.

The coefficients of partial correlation are given in Table 19. Here again in all essential respects this series of coefficients agrees with the previous series.

TABLE/

TABLE 20. Coefficients of multiple correlation in county boroughs.

$$R_{1.459} = .508 \pm .067$$

$$R_{2.459} = .405 \pm .062$$

$$R_{3.459} = .594 \pm .048$$

The total correlations (Table 20) are also of the same order of magnitude and show the same differences in the three sections of the infant death rate as those already given for counties. Consequently it would appear that all of these findings give support to the conclusions previously reached.

(c) Urban and rural districts. In urban and rural districts, returns of still-births are not available, but the statistical constants have been calculated for the neo-natal and post-natal death rates. As will be seen from Table 21,

TABLE 21. Means, standard deviations and coefficients of variation of the infant death rates in urban and rural districts.

	<u>Urban districts</u>		<u>Rural districts</u>	
	Neo-natal mortality	Post-natal mortality	Neo-natal mortality	Post-natal mortality
Mean	30.81	38.08	29.53	31.67
Standard deviation	4.27	10.18	4.02	9.42
Coefficient of variation	13.84	26.75	13.71	29.74

the rates of mortality in both periods of infant life are higher in urban than in rural districts, but that each of the death rates of these two aggregates of districts is lower than in county boroughs. Here again, however, it will be seen that the post-natal death rate varies more from town to country than does the neo-natal death rate. Comparing county boroughs and rural districts, the neo-natal rate is 13 per cent. higher, but the post-natal death rate in county boroughs is 56 per cent. higher than/

than in rural districts. Similarly the aggregate of urban districts shows a neo-natal death rate 5 per cent., and a post-natal rate 20 per cent. higher than in rural districts. The coefficients of variation of the neo-natal mortality rates tend to be slightly greater in towns than in rural districts, whereas the variability in the post-natal death rate tends to diminish in passing from rural districts to large towns.

TABLE 22. Coefficients of correlations between the infant death rates and certain influential factors in urban and rural districts.

	Urban districts.		Rural districts	
	Neo-natal mortality	Post-natal mortality	Neo-natal mortality	Post-natal mortality
Death rate in females 15-45	.566 \pm .070	.772 \pm .042	.425 \pm .084	.587 \pm .067
Maternal mortality rate	.308 \pm .093	.341 \pm .091	.383 \pm .088	.528 \pm .074
Puerperal sepsis death rate	.043 \pm .103	.151 \pm .100	.180 \pm .100	.372 \pm .089
Maternal death rate less sepsis	.440 \pm .083	.377 \pm .088	.581 \pm .088	.456 \pm .065
Rooms per person	-.529 \pm .074	-.775 \pm .042	-.516 \pm .075	-.745 \pm .046
Domestic servants per 100 population.	-.576 \pm .069	-.648 \pm .060	-.497 \pm .077	-.629 \pm .062

The coefficients of correlation are given in Table 22.

From these it appears that there are no great differences from the results already found.

TABLE 23. Coefficients of partial correlation.

(a) Urban districts.

$r_{24.59} = .229\pm.097$	$r_{34.59} = .552\pm.071$
$r_{25.49} = .142\pm.101$	$r_{35.49} = .084\pm.102$
$r_{29.45} = -.323\pm.092$	$r_{39.45} = -.260\pm.096$
(b) Rural districts	
$r_{24.59} = .077\pm.102$	$r_{34.59} = .198\pm.099$
$r_{25.49} = .273\pm.095$	$r_{35.49} = .437\pm.083$
$r_{29.45} = -.341\pm.091$	$r_{39.45} = -.454\pm.082$

The/

The coefficients of partial correlation, however, (Table 23) do show some distinct differences. Consider first the neo-natal death rates. In both urban and rural districts there is a positive and significant correlation with the index of environment, and these are of the same order of magnitude as already found both for counties and county boroughs. Also in both aggregates of districts the health of the mother shows no significant relationship with the death rate at this period of life. With the total maternal mortality rate, however, the coefficient for rural districts is barely significant, and that for urban districts, although positive, is, with regard to its probable error, quite insignificant. These results, therefore, contrast with the previous findings. With the post-natal death rates, differences are also noticeable. In urban districts all the coefficients agree with what has already been found. The index of maternal health shows the highest correlation, environment a smaller but apparently significant correlation, and the mortality rate of women in childbed no relationship. In rural districts, on the other hand, environment would seem to play a larger part than the health of the mother, which shows a positive but statistically insignificant correlation, whereas a definitely significant positive correlation is found with the maternal mortality rate in childbearing. A result similar to this was found for counties, but when, instead of the total maternal mortality, the death rate from causes in childbed other than puerperal sepsis was taken as the index of medical care of women in pregnancy and at childbirth, the final correlation became insignificant. Consequently all the partial correlations have been recalculated in county boroughs and in urban and rural/

rural districts. There are given in Table 24.

TABLE 24. Coefficients of partial correlation.

	County boroughs	Urban districts	Rural districts
$r_{24.79}$.191 \pm .072	.180 \pm .100	.083 \pm .102
$r_{27.49}$.221 \pm .071	.378 \pm .088	.285 \pm .095
$r_{29.47}$	-.252 \pm .070	-.366 \pm .089	-.342 \pm .091
$r_{34.79}$.498 \pm .056	.540 \pm .073	.236 \pm .097
$r_{37.49}$.036 \pm .074	.241 \pm .097	.324 \pm .092
$r_{39.47}$	-.391 \pm .063	-.282 \pm .095	-.436 \pm .083

In county boroughs, it will be seen that no difference is made in any of the coefficients by substituting this second index of medical care. In urban districts, the neo-natal death rate now shows a significant positive correlation with this index, and the remaining two factors, environment and health of the mother, show no significant changes in their relationships. With the post-natal death rate, the correlation with this second index of the quality of obstetrical supervision is greater than that found by using the total maternal mortality rate, although it still does not reach the customary standard of significance. In rural districts the correlations involving the neo-natal death rate do not differ substantially from the results already found; but those involving the post-natal death rate show some slight change. The correlation with the measure of maternal health has been raised, but not to any significant degree; that with environment remains unchanged; but the association with the death rate of females from childbed causes other than sepsis, although somewhat lower than that which was found with the total maternal mortality rate, is still quite significant with regard to its probable error. Comparing the correlations with this factor/

factor in the three aggregates of districts, there seems to be a definite tendency for its value to increase as we pass from highly urbanised communities to scattered rural districts. A series of results such as this would, therefore, lead to the suspicion that this index of the medical supervision of pregnant and parturient women was also indicative of some other factor influential in infant life. It has been suggested previously that, as this death rate is dependent on the proximity to medical care, a relationship such as this might arise where, in any group of districts, a part of the death rates depended on the rapidity with which competent medical skill could be summoned. The trend of this series of coefficients seems to be sufficiently definite to justify the assumption that some such explanation may suffice. If it be true, then it leads to the conclusion that in rural communities there is some portion of the post-natal death rate of infancy, due presumably to acute conditions, which could be prevented by the timely arrival of medical assistance, and that deaths such as these form an insignificant part of the death rates in urban communities.

TABLE 25. Coefficients of multiple correlation.

	Urban	Rural
$R_{2.459}$.632 \pm .062	.568 \pm .070
$R_{3.459}$.790 \pm .039	.749 \pm .045

The coefficients of multiple correlation for the two death rates, neo-natal and post-natal, in urban and rural districts are given in Table 25. These agree very well both with one another and with those already found in counties and county boroughs, the coefficients being in both instances higher for the post-natal than for the neo-natal mortality rates/

rates.

These results for widely different parts of the country are so generally consistent one with another, both with regard to sign and magnitude, that the inferences to be drawn from them may be stated with a reasonable degree of assurance.

(1) Ante-natal deaths. The ante-natal death rate is definitely associated with the rates of mortality of mothers from causes of death peculiar to childbearing. A reservation must, however, be made in that the main single cause of maternal death, puerperal sepsis, has no significant relationship with the death rate at this period of infant life. The size of the correlation between the two rates of mortality, however, is not of the order of magnitude which would lead to the belief that this was the only factor of importance in determining the height of the still-birth rate. Although the results seem to justify the hope that the increased attention to mothers during pregnancy and both at and after labour will be the means of reducing some causes of death of infants in ante-natal life, the actual saving of infant life at this stage will, in proportion to the total, not be very great. Further, so far as our data can be trusted, the health of the mother and differences in environment or social status do not, when allowance has been made for variations in the child-bearing mortality rates, appear to have any influence on the still-birth rate. Although there is a significant correlation with maternal health, the partial correlation coefficients show that this is a secondary association due to the fact that both the ante-natal death rate and the general mortality of women of reproductive ages are related to the mortality rates of mothers in childbearing. These results are in accord with the findings of Bruce Murray (1924). The comparison of the weights and lengths/

lengths of the offspring of primiparous women in the pre-war, war and post-war periods made by this author shows that the health and nutrition of the mother during pregnancy has no effect on the state of the nutrition of the infant at birth, and that "the foetus lives, like a true parasite, regardless of the expense of the mother". It seems not unreasonable to conclude, then, that external factors acting on the mother do not affect the infant's chance of survival prior to birth. The coefficients of multiple correlation show that the sum total effect of these three factors on the ante-natal death rate is not very large, and leads to the conclusion that a large part of this death rate would still remain, even if it were possible to remove the conditions which these indices measure, and which are prejudicial to the survival of the foetus.

(2) Neo-natal deaths. The neo-natal death rate is also significantly associated with the death rates of mothers in childbearing, so that it seems justifiable to infer that some part of this early mortality is within the scope of an energetic obstetrical service. Further, our results show that at this stage of life, changes in environment seem to be factors of importance in determining the height of the mortality rate. This finding would appear to be contrary to accepted opinion. Brend (1917), comparing the average death rates from neo-natal causes in various groups of social class, concludes that "the great bulk of these deaths are due to some obscure internal derangement of normal processes in the mother or infant which are either independent of external environment or are due to some factor or factors in the external environment equally common among all classes under all circumstances." The figures quoted by Brend do, however, show a rise in the neo-natal mortality/

mortality rates with descent in the social scale although the differences are not very great. But, as the present analysis shows, the variations exhibited in the mortality at this period of life are not nearly so wide as those shown by the post-natal death rate. Consequently, provided the correlations with social status were equal, a simple comparison of the mean values in different groups of social classes will not show such striking differences with the neo-natal as with the post-natal mortality rates. Our results show that, in spite of the low variability in the neo-natal mortality, these smaller variations are almost as intimately connected with changes in the external environment as are the wider variations in the post-natal death rates. The only difference between the two is that for equal changes in environment the neo-natal death rate will not show a reaction as extensive as will the post-natal death rate. That is, although the correlations are almost **equal**, the coefficient of regression on environment of the neo-natal rate is lower than that of the post-natal rate. That a relation such as this should exist so early in infancy is by no means absurd. Even if it be true that at birth infants of all social classes are equally likely to survive if they could be placed under similar circumstances after birth, it must be remembered that the infant has suffered a sudden and complete change in its mode of existence. In utero, all the functions characteristic of extra-uterine life had been performed for it by the mother. Its food is pre-digested and conveyed to the foetus in a form immediately available for assimilation, and aeration of its blood and the excretory processes are also carried out by the **p**lacental circulation and not by the foetal organs themselves. But examination of the amniotic fluid has revealed traces of foetal urinary constituents, and/

and in obstructed delivery meconium may be passed from the foetal bowel, so that in all probability, at term, foetal development has reached a stage when the organs are capable of performing the functions required of them in after-life. A comparison of the extra and intra-uterine environments, however, shows that immediately after birth a sudden difference occurs. In utero, the foetus lies bathed in a fluid of almost uniform (body) temperature, whereas at birth it is expelled into a much colder atmosphere. And it is obvious that the heat-regulating mechanism can have had no previous trial of its efficiency, as seems to be possible with the heart, kidneys, and bowel. Thus it does not seem unreasonable to suppose that one of the important factors at this stage of life is the adequate conservation of the body heat of the new-born child. In this connection, some results recorded by Louise McIlroy (1925) are of extreme interest and importance in demonstrating the sensitiveness of the new-born to changes in environment. A series of babies at birth were cleaned with olive oil instead of by bathing, and it was found that these lost less weight after birth than did the infants who were bathed in the ordinary manner. Even better results were found when, in addition to being oiled, the infant was transferred to a cot with warm blankets and hot water bottles. These results show that the loss of heat is an important consideration to the infant at birth. Thus it does not seem improbable that, as our results show, differences in the environment into which an infant is born may be of no small importance. The health and nutrition of the mother do not seem to be factors of such great moment in determining the fate of the infant in this early stage of life. The coefficients of multiple correlation show that, together, these three factors can only/

only account for 19 per cent. of the variability in the neonatal rates of mortality. Here again we must recognise the existence of other factors than these determining the chance of survival.

(3) Post-natal deaths. The causes of death in post-natal life seem to be those which are most amenable to control by administrative measures or organised medical effort. At this stage, the health of the mother takes precedence over environment, whereas the effects of the obstetrical aid afforded to the mothers in childbearing have by this time worn off. It has also been suggested, from a comparison of the results found for large cities and scattered rural communities, that in the latter there is a portion of the post-natal death rate, and this most probably due to urgent conditions, which could be removed if medical assistance were more readily available.

IV. THE CAUSES OF STILL-BIRTH.

In the ~~official~~ reports of this country there is no record of the causes of death among still-born infants. Table 26 has, however, been prepared from the admirable statistical returns of the Netherlands to show the causes of ante-natal deaths in that country. The figures are given separately for males and females, and the rates of mortality per 1000 births and the percentage frequency of each of the causes of death are given together. The births and deaths on which these rates and frequencies are based relate to the twelve years 1911-22.

TABLE 26. Showing the proportional frequency of, and the mortality rate per 10,000 live-births from several causes of still-birth in the Netherlands 1911-22.

	<u>Mortality</u>		<u>Frequency</u>	
	Male	Female	Male	Female
Syphilis	0.58	0.64	1.44	1.81
Other general diseases of the mother	0.85	0.79	2.05	2.21
Habit abortion	0.56	0.56	0.39	1.01
Albuminuria	1.42	1.46	3.52	4.08
Traumatism and prolonged labour	0.23	0.25	0.70	0.69
Placenta praevia	2.84	2.50	7.02	7.01
Foetal deformities	2.48	2.95	6.14	8.27
Premature birth	7.11	6.04	17.59	16.93
Difficult labour	4.76	3.32	11.77	9.31
Torsion and compression of the umbilical cord	2.80	2.04	6.92	5.71
Foetal asphyxia	2.30	1.93	5.69	5.41
Unclassified causes	1.74	1.52	4.31	4.26
Unknown causes	12.91	11.90	31.95	33.33
All causes	40.42	35.70	100.00	100.00

A grave objection which is most obvious in these figures is the large proportion of foetal deaths in which the death is registered from an unknown cause. The defect is one which prevents accurate comparison with any figures collected from a more accurate source, such as a hospital, in which the investigator/

investigator in all probability will combine clinical examination of the pregnant woman with subsequent postmortem examination of the dead-born foetus. The proportion of deaths from unknown causes in this series is probably the result of the two factors: (1) that many of the women have not been seen by either a doctor or midwife before the infant was born, and (2) that postmortem examinations are seldom, if ever, carried out in general practice. If the defect in these figures were solely due to the fact that in all these cases the mother had not been attended until after the child was born, by distributing these deaths from unknown causes to each of the known cause groups according to the proportional frequency of each of these, a fair degree of comparability between these figures and more accurate statistics might be obtained. But since this is not the sole cause of such a proportion of unknown deaths, this procedure must obviously be wholly inaccurate, since it would amount to the assumption that all causes of foetal death were equally easy to diagnose - an extremely improbable assumption. On the other hand, these figures are of some value as a general guide to the most important causes of foetal death in a random sample of the total population. More accurate investigations, due to the selected nature of the material, normally afford a biased view of the relative importance of certain causes of death. The type of selection will depend on the source of the data. Hospital statistics would be weighted in favour of foetal deaths from causes which at the same time endanger the life of the mother. Consequently these causes which endanger the lives of both mother and foetus in utero will be over-represented as causes of foetal death as compared with the frequency pertaining/

pertaining to the general population; whereas foetal deaths due to conditions proper to the foetus, which cause neither disease in the mother, nor difficulties in delivery from disproportion between the presenting part and the maternal pelvis, will be under-represented. That this is the case can be shown by a comparison of the proportion of foetal deaths from a complication such as torsion of, or pressure on the umbilical cord. This condition will of itself cause no difficulty to the mother, and consequently should be under-represented in a hospital population of still-births. In a recent report by Holland and Lane-Clayton (1926), in 41 out of 1673 dead-born infants, the death was due primarily to this complication. In the figures quoted here for the Netherlands, 5219 of the total 81,773 still-births were due to the same cause. In the former case the proportion, therefore, is 2.45 per cent., in the latter 6.38 per cent., that is more than double the proportion from this cause are found in the general population as compared with a selected hospital population. The group comprising foetal deformities does not offer the same grounds for comparison, because, although many of these are due to hydrocephalus and the obstruction resulting from the excessive size of the head in many instances causes interference with labour, other deformities, such as congenital cardiac malformations, will cause no obstruction to delivery; so that congenital malformations will only be included under causes of foetal death which also endanger the life of the mother provided that an excessive proportion of deformities are those causing obstruction to delivery. Actually in the Netherlands statistics, the proportion due to foetal deformities is 7.08 per/

TABLE 27. Percentage frequency distribution of causes of dead-births.

Cause of death	Dutch	Holland and Lane-Claypon
(Syphilis	2.7	9.5
(Other general diseases	4.3	2.7
(Habitual abortion	1.4	-
(Albuminuria	7.7	12.1
(Precipitate labour	1.2	-
Diseases of father		
or mother		
Placenta praevia -----	12.3	22.1
Deformities, including hydrocephalus -----	10.4	11.5
Premature labour not due to disease of father		
or mother -----	22.5	3.9
Pelvic deformities and malpresentations -----	15.5	33.0
Pressure or torsion of cord -----	11.2	3.4
Asphyxia at birth -----	7.4	0.7
Other causes -----	5.7	1.1

per cent. whereas in the Report by Holland and Lane-Clayton 8.25 per cent. of the total still-births were from this cause.

Table 27 has been quoted from these authors to show the differences between the Dutch figures and those collected in their extensive special enquiry. (The figures for Holland relate to the years 1901-22.) The enormous excess in the hospital series of such causes as placenta praevia, pelvic malformations and malpresentations, which are conditions obviously endangering both mother and child, the corresponding defect of such conditions as pressure and torsion of the cord, premature labour and asphyxia at birth, which, as a rule, only endanger the child, support the suggestion that the data are not really in pari materia. We should expect such a difference of classification if the hospital data really included an over-average proportion of labours in which the mother's life was jeopardised, and need not attribute the whole of it to more accurate diagnosis. If we are to try to measure the advantage to be gained by the foetus by careful supervision of the parturient during pregnancy and at labour, it is surely better to err on the side of under rather than over-statement. Hence it is better to estimate the proportion of avoidable causes of foetal mortality on the basis of general population statistics, faulty, no doubt, but not open to the suspicion - as institutional records clearly are - that the relative frequency of factors endangering both mother and child is over-stated.

The total still-birth rate in Holland for the twelve years 1911-22 (irrespective of sex) is 3.90 per 1000 live-births compared with a rate of 3.11 per 1000 live-births for England and Wales (1921-3). In view of the small difference/

difference in the ante-natal loss of life in the two countries and in spite of the fact that there may be some international differences in the frequency of the several causes of death, probably something of value is to be gained from these figures and possibly the amelioration likely to be effected by intensive prophylactic efforts on the part of our national obstetrical services will be more conservatively judged from these than from the records of lying-in institutions. Table 26 shows that the ante-natal mortality of males is 13 per cent. greater than that of females. For individual cause groups, the greatest proportional excess in the male death rate is in the group "difficult labour", and this is followed closely by the death rate from pressure on or torsion of the umbilical cord. The former group shows a death rate 43 per cent. higher in males than in females, the latter 37 per cent. excess in males. These figures might be taken as evidence in support of the view that the greater size of the male head is a cause of some greater difficulty in labour than there is with a female birth. The other group of causes of death in which such a factor might be revealed is the relatively unimportant cause, "traumatism and prolonged labour" in which, however, the male death rate is only 13 per cent. in excess of the female. The sole cause of death in which females have a fairly large excess mortality is from foetal deformities, in which the male death rate is 16 per cent. lower than the female. The doubtful figures for deaths from syphilis give a rate 9 per cent. lower in males than in females; but, as in this country, probably little reliance can be placed on these figures.

With regard to the relative importance of the causes/

causes of death, leaving out of consideration the deaths from unknown causes, premature birth claims the greatest number of the deaths both in males and females. Difficult labour is the second largest cause of death, and is followed closely by placenta praevia, and foetal deformities. Torsion and compression of the cord accounts for 6.4 per cent. of deaths, and albuminuria for only 2.1 per cent. It will be seen then that many of the causes of foetal death are also important causes of maternal death, and gives reason for the belief that the further extension of care to pregnant women will result in benefit for both the mother and child. But if, as seems probable, the majority of the group of unknown causes is not associated with maternal distress (if so they would have been included under difficult labour or some similar category), and noting the large proportion of deaths from premature birth and foetal deformities, the latter being entirely, the former in great measure, outwith human control, it would appear that even with the best obstetrical services in the world, a high ante-natal loss of life will always remain. Hospital statistics in this connection are apt to produce a too optimistic outlook, since, as has been shown, these contain an undue proportion of foetal deaths from causes in which obstetrical supervision would benefit both mother and child. This results from the selected population studied. But the figures given here afford reason for some scepticism as to the advantages to the foetus of increasing attention to the mother.

V - VISCERAL VARIABILITY IN FOETAL AND INFANT LIFE, AND ITS BEARING ON THE PROBLEM OF MORTALITY.

In most statistical investigations of the mortalities in infancy probably more consideration is given to the effects of external factors acting on the infant than to the changes which take place in the infant itself as growth proceeds. Brownlee (1917), from a study of the mortality rates at ages from various causes of deaths in infancy and childhood, has shown that these obey certain definite laws, and his results serve to emphasise the importance of further exploration of the changes in the physiological processes of the developing child.

The biometric constants relating to man have in the past been practically always confined to the adult period of life. Visceral and skeletal measurements and interrelationships at this period are fairly well known; but until recent years little interest seems to have been taken in the foetal and early infant stages of life. Signs are not wanting, however, that the defect is being appreciated. Holland (1922), in a part of his investigation into the causes of foetal death, published data with regard to the weights of five viscera, in addition to body weight and length and placental weight in a series of dead-born infants collected from several lying-in hospitals. The total series of 300 foetuses is divided into three sets: (1) foetuses examined in the fresh state, (2) those born in a macerated condition, and (3) a small group of syphilitic foetuses. Since the autolytic processes associated with maceration affect the organ weights to varying degrees, depending for the most part on the enzyme content of the organ, and since such data can throw/

throw no light on the anatomical differences in health and disease, the short series of foetuses born and examined in the fresh state is only considered here.

(1) Visceral variability in foetal life. A difficulty which confronts any investigation of this kind is that analysis can only be carried out on the statistics of dead infants, and it is essential that the statistical constants be compared with some control series. Since we obviously cannot know what is the degree of variation in the viscera of living healthy foetuses and infants, an attempt must be made to obtain the closest approximation to this. From the already short series of 143 fresh foetuses, 71 of these, in which the cause of death was prolonged labour, instrumental delivery or torsion of or pressure on the cord, have been extracted as a presumably normal series. This selection is, of course, open to the objection that most of these causes of death are probably associated with ~~most~~ *some* pelvic deformity of the mother causing dystocia, so that the selection may be one of a series of foetuses from mothers whose general health is below the average. But it is the nearest approach to normality which can be extracted from the data, and, if our previous results can be trusted, the health of the mother does not appear to be of any significant importance in the life of the infant at this stage.

The analysis of these data will be confined to a discussion of (a) the relative variabilities in the foetal organs of the "normal" and total series, (b) a comparison of foetal with adult visceral variability, and (c) the differences in the variation of the sexes.

TABLE/

TABLE 28. Showing the coefficients of variations for certain body characteristics of the foetus
(a) "Normal" series, (b) Total series.

	(a) "Normal"	(b) Total
Body weight	25.56	31.51
Body length	9.05	10.67
Thymus	43.79	47.32
Liver	33.59	37.62
Spleen	35.43	56.48
Suprarenals	30.65	41.47
Kidneys	30.24	34.79

(a) Table 28 contains the coefficients of variation in the two series of foetuses. In the normal series the thymus is the most variable organ and the kidney the least variable. Body weight and body length show less variation than does any of the viscera. In the total series, the spleen takes precedence over the thymus in variability, but the other organ weights show no change in their position relative one to another. Comparing the two series, it will be seen that in every instance, the variation is greater in the total than in the normal series. The excess is more marked with body weight than with body length. Among the viscera, the spleen would appear to be the organ which suffers the greatest change in disease at this period of life. The suprarenal glands are also greatly affected, and the liver, kidneys, and thymus show the smallest changes. There is, however, an obvious objection to the above comparison. Our normal series consists of foetuses which have survived until full time and consequently are all of approximately the same age, whereas the total series consists of foetuses who have reached a stage of viability but whose ages differ more widely (although not to a very great degree). So that we have been comparing two groups of foetuses in one of which differences in age are so relatively small as to be negligible, whereas in the other, differences in age may be of great/

great importance, since in the few months preceding full time, the foetus is growing very rapidly. I have therefore proceeded a stage further and calculated in both series the coefficients of variation for constant body weight and length. This will remove as far as possible the limitations of the previous comparison. The new coefficients are collected in Table 29.

TABLE 29. Showing the visceral coefficients of variations for constant body weight and length.

	(a) "Normal"	(b) Total
Thymus	35.46	40.59
Liver	19.61	20.16
Spleen	28.72	52.88
Suprarenals	29.41	33.63
Kidneys	26.80	26.78

Except in kidney weights, the normal series still shows a less degree of variability than does the total series. It will also be seen that the liver is not affected to any significant extent in passing from a normal to a diseased population of foetuses. Further, the spleen still shows the greatest reaction in disease. The two ductless glands, thymus and suprarenals, are affected approximately to the same degree.

TABLE 30. Showing the coefficients of variation in "healthy" and "hospital" populations (adults)

	(a) Healthy	(b) Hospital
Liver	14.80	21.12
Spleen	38.21	50.58
Kidneys	16.80	24.63
Body weight	10.37	-
Body length	3.99	-
Heart	17.71	32.59

(b) The variability in the viscera of foetuses may now be compared with the following table (Table 50) from Pearl (1905) showing the coefficients of variation in the adult "healthy" and "hospital" populations. In all the comparable data it will be noted that the foetal organs show

a/

a much wider range relatively than do all the organs of healthy adults, except the spleen. From this table also we see that, in comparing a healthy and a diseased population, the differences in variability produced by disease in adult life are much greater than are those found in the two foetal series given here. Since chronic diseases affect the organs to a greater extent than acutely fatal illnesses, we must conclude either that diseases affecting the foetus in utero are rapidly fatal conditions which allow but little time for the production of gross changes in the organs, or that the foetus is already in such an unstable condition physically that any untoward circumstance of however slight a character rapidly upsets the normal processes.

The results of the above comparison, therefore, lead to the conclusions that the foetal viscera are extremely variable both in health and disease; that disease affects the foetal organs to a less extent than does disease in adult life, and that to judge by analogy, foetal death from disease occurs very rapidly.

TABLE 31. Showing the coefficients of variation for the sexes in the total series of foetuses.

	<u>Male</u>	<u>Female</u>
Body weight	34.73	24.38
Body length	11.50	8.99
Thymus	53.07	41.06
Liver	38.43	31.93
Spleen	63.42	34.89
Suprarenals	40.54	40.35
Kidneys	35.38	33.75

(c) The coefficients of variation for the sexes are given in Table 31. These show that the male foetus in every instance exceeds the female in variability and, although here again the differences are small, the uniformity in the series is sufficiently striking to warrant the conclusion that the male at this stage of life is really the more variable. Pearson (1897)/

(1897) has shown that in adult life the female is slightly more variable than the male, and this he attributes to a relatively less intense struggle for existence. This explanation is obviously insufficient to account for differences in variability in intra-uterine life where any struggle for existence must be shared equally by the two sexes. The extremely high variability of both sexes in foetal life, however, can probably be explained on this assumption. Before birth, the foetus is a parasite, not depending on its own organs for carrying out the functions required of them in post-natal life, so that the organism has little need of them at this early stage, and it would appear "that each organ has a life and growth of its own, irrespective of the needs of the organism as a whole". The lack of any struggle for existence, therefore, would appear to be reflected on the physical characters of the foetal viscera.

(2) Visceral variability in infancy. From data collected by Professor Turnbull in the Pathological Department of the London Hospital, and to which I have been allowed access, a series of coefficients of variation have been calculated for certain organ weights and for body weight and length in the first year of life to show the changes that occur during this period. The data available are insufficient to extract from them a "healthy" series at each of the ages under one year, so that the vast majority of these infants have died from some disease processes. Still the short comparison which it has been possible to make reveals certain points of interest.

TABLE/

TABLE 32. Showing the coefficients of variation for certain characters at several age periods in infant life.

(a) <u>Males</u>	Under 1 week	1 week-1 month	1-3 months	3-6 months	6-9 months	9-12 months
Body weight	31.34	34.45	30.44	28.79	29.59	27.89
Body length	9.35	8.10	9.19	8.56	8.62	9.27
Heart	40.35	47.31	40.35	35.58	32.67	32.67
Spleen	70.31	91.21	82.83	63.37	76.89	52.02
Kidneys	40.48	50.86	42.64	36.86	31.91	29.03
Thymus	61.88	74.77	102.51	98.98	85.42	78.79
(b) <u>Females</u>						
Body weight	32.93	28.96	26.84	31.90	33.64	25.43
Body length	9.48	9.36	9.16	9.18	9.56	8.47
Heart	51.90	43.66	46.91	42.21	33.90	31.90
Spleen	52.68	73.79	105.45	76.54	59.99	57.02
Kidneys	52.84	45.49	49.71	42.66	33.97	26.25
Thymus	59.05	74.43	87.61	83.91	85.22	88.01

The coefficients of variation are given in Table 32.

In nearly every instance the coefficients are lower both in males and females in the first age period (foetuses and deaths under one week) than in the immediately subsequent group. The values given for this early age compare reasonably well with the values given already for male and female foetuses. After the first week, the variability shows a general tendency to decline as the infant grows up. Irregularities in some of the age groups are apparent, especially with the thymus and less so with the spleen, but the variability at the end of the first year of life has become definitely less than it was in the first month with the other organs. And further, when it is remembered that the older the infant the greater is the chance of death from some chronic condition, and therefore that, other things being equal the variability should increase with age in this set of data, it would be even more likely that the decrease as represented by these figures is smaller than what actually does occur. With regard to the lower variability in the first week of life as compared/

compared with later ages, it is in all probability not a real phenomenon because all of these deaths are from accidents of birth, and consequently these cases really represent a more or less "healthy" population, so that the series of foetal deaths and deaths under one week are not really comparable with those occurring later in infancy, which are due chiefly to broncho-pneumonia or gastro-enteritis. In this series no constant differences in the sexes can be demonstrated.

From the foregoing analysis, then, it may be concluded that the variations in infant viscera tend to become smaller as age advances. Under a given environment, high variability is likely to be indicative of instability, and Greenwood (1904), from his comparison of the viscera in diseased and normal adults, has shown that this is typical of the diseased state in adult life. To this it seems justifiable now to add that high variability is also characteristic of certain phases of life, diseased conditions entirely apart. Instability in this sense is apparently present at puberty. It is also a feature in ante-natal life in which the male is probably more unstable than the female. In infancy, too, instability is evident, and, as the infant continues to grow, part of this wears off gradually.

VI. - INFANT MORTALITY AND ECONOMIC STATUS:
A NEW METHOD OF ASSESSING AND ELIMINATING
THE INFLUENCE OF THIS FACTOR ON RATES OF
MORTALITY.

When we desire to measure the effect upon the death rate of some possible or probable factor we always seek to neutralise any other variable factor the influence of which, however intrinsically important, is not relevant to the object of the immediate inquiry. Thus if we are for the moment interested in the influence of occupation upon mortality we should seek to allow for the fact that the age distributions of workers in different occupations vary much, because we know that age is a predominant factor of mortality and that variations of age might mask any other difference. Many expedients have been devised for this end, and each has its advantages and disadvantages.

A much used method, rejoicing in the formidable title of the method of Indirect Standardisation, may be simply illustrated. Let us suppose a simple dichotomy of the population into A's and B's, and that the average death rate of the A's is twice that of the B's, say 20 per 1,000 and 10 per 1,000 respectively for all the A's and B's of the population, without regard to their occupations, place of residence or any other matters. We now wish to study the effect of, say, occupation upon mortality and we find that the proportions of A's ~~to~~ B's vary from occupation to occupation and that in each occupation we can measure the ratio of all deaths to population but not distinguish between the deaths of A's and deaths of B's although we can distinguish between the enumerated A's and B's at a recent census. Then we might proceed/

proceed in this way. We might take as a 'standard' a population with 50 per cent. each of A's and B's which, by hypothesis, would have a death rate of 15 per 1,000. Now suppose we have two occupations in one of which the proportion of A's is 25 per cent. and in the other it is 75 per cent. and that in one the death rate is 12 per 1,000 and in the other 18 per 1,000. We should expect the former group to have a lower death rate, whatever the effect of occupation on mortality may be, because it contains a much smaller proportion of A's who, under average conditions, have a higher rate of mortality than B's. Is it reasonable to attribute all the difference to this circumstance? Let us suppose that in each district the rates of mortality were exactly average and the gross difference wholly due to a change in the proportions of A's. In the first group we should expect a death rate of $0.25 \times 20 - 0.75 \times 10 = 12.5$ per 1,000 instead of 15 per 1,000, and in the second group we should expect a death rate of $0.75 \times 20 - 0.25 \times 10 = 17.5$ per 1,000 instead of 15 per 1,000 the gross rate when A's and B's are equal in number. We thus see that the group rates actually found, 12 and 18, are not exactly what they would have been had the assumption been correct, but we also see that this factor might so nearly account for the observed difference that it would not be sensible to compare 12 and 18 as if the varying proportions of A's could be neglected. What shall we do? An obvious suggestion is to multiply the observed rates by factors which would make the resultants exactly the same if the differences were entirely due to varying proportions of A's. The required factors are $15/12.5$ and $15/17.5$ respectively, i.e. 1.2 and 0.8571. Multiplying the observed rates 12 and 18 by these factors we reach 14.4 and 15.4 as values which, on our hypothesis are reasonably comparable as measures/

measures of contrasting factors other than the varying proportions of A's.

The most serious assumption involved in this process - from the point of view of medical-statistical interpretation - is that, in different groups the relative mortalities of A's and B's should be the same. It is easy to imagine cases where the assumption is absurd. Thus suppose our A's were soldiers and our B's civilians and that a 'little war' was on, and one of our groups was in the fighting zone. The method of correction would go a very little way towards freeing the geographical comparison from the effects of varying proportions of A's and B's, because if it was really a very little war, its effect upon the standard rates of mortality for all soldiers and civilians would be very small. Nevertheless, the method is a valuable one and has been widely used to eliminate variations of age constitution in population groups it is desired to compare. The object of the present section of the study is to illustrate its use in another demographic field.

We know that economic status is a factor of mortality - whether directly or indirectly is a question which does not now arise - and we know that the rate of Infant Mortality seems to be particularly sensitive to variations of economic well-being. We know also that the proportions of 'rich' and 'poor' vary greatly in different administrative areas. If we adjust the local rates of infant mortality by the method described above, do we appreciably reduce the inter-local variation? The recent publication of the Registrar-General's Decennial Supplement (Part II) relating to occupational mortality, fertility and infant mortality has made it possible to answer that question. The objects of the present note are (1) to describe a simple method/

TABLE 33. Relative mortalities in different social classes.

	I	II	III	IV	V
Infectious diseases	.22	.57	.96	1.19	1.34
Tuberculosis (all forms)	.38	.68	.93	1.26	1.11
Diarrhoea and Enteritis	.32	.59	.94	1.13	1.42
Developmental and wasting dis.	.64	.81	.98	1.09	1.14
Bronchitis	.14	.53	.98	1.24	1.24
Pneumonia	.28	.53	.97	1.16	1.36
Bronchitis and Pneumonia	.23	.53	.98	1.19	1.32
Syphilis	.22	.47	.93	1.21	1.44
Rickets	.36	.59	.86	1.14	1.50
Convulsions	.54	.63	1.01	1.16	1.13
Congenital Malformations	.98	.96	1.00	1.02	1.01
Congenital debility and sclerema	.34	.67	.96	1.15	1.25
Premature birth	.63	.81	.99	1.09	1.12
Injury at birth	1.32	1.16	.97	1.04	.86
Suffocation in bed	.45	.60	.91	1.08	1.60
Infant mortality	.48	.70	.97	1.13	1.23

method by which such a correcting factor for differences in social position may be obtained, and (2) to ascertain (a) how far, when such correction is made, the variation observed in the rates of infant mortality in a sample of districts will be reduced, and (b) whether the position of the several districts in respect of mortality is altered by such a correction.

(1) Method of correcting for variations in social status: From Table H of the report referred to previously, the number of births and the number of deaths from various causes or cause groups of infants under one year of age can be obtained for each of the five social classes recognised in these official reports. (These are classes I, III and V consisting of the upper and middle classes, skilled workers and unskilled workers respectively, and groups II and IV intermediate between I and III and III and V respectively). From these data the rates of mortality per million births are calculated, and comparative mortality figures (C.M.F.) for each disease group then obtained by expressing the death rates pertaining to the legitimate births of each of the classes as a fraction of that of all legitimate births in England and Wales. (The data refer to the single year, 1921.) In Table 33, these are presented for all the causes of infant death supplied by the report. The varying degrees to which economic conditions affect the several causes of death in infancy are thus clearly demonstrated. They range from cases where the higher the social class the higher the rate of mortality, e.g. injury at birth, through a point of indifference in the group congenital malformations, to an immense advantage for the more prosperous classes in such groups as the common infectious diseases, bronchitis and pneumonia, syphilis and infantile diarrhoea/

diarrhoea. Discussion of the reasons for these differences will not be attempted here.

The male inhabitants of the district to be considered must now be distributed into the five social groups of occupation. Information relative to the occupations of the male population is obtained from the Census reports, in which the occupations of males aged 12 years and over are given for individual districts; and combined with table A, column 7 of the Occupational Mortality Report, which gives the recognised social rank of each occupation, we are enabled to obtain the social distribution of each district. The further procedure, which is only an elaboration of my hypothetical example of A's and B's is made more intelligible by reference to the example given on Table 34, where the case of the Metropolitan Borough of Hampstead is considered.

TABLE 34. Example of method of correction for social status. Metropolitan Borough of Hampstead.

Social class	Population	Weights	Weighted population.
I	2686	.4847	1301.9042
II	8523	.6998	5964.3954
III	8433	.9708	8186.7564
IV	1789	1.1295	2020.6755
V	1603	1.2264	1965.9192
	23034		19439.6507

$$\text{Correcting factor} = \frac{23034.0000}{19439.6507} = 1.1849$$

Crude infant mortality rate (1921-3) = 55.3 per 1,000 births.

Corrected infant mortality rate (1921-3) = 55.3 x 1.1849 = 65.5 per 1,000 births.

The comparative mortality figures for total infant mortality are shown in the third column of this table. The adult male population/

population of the borough, enumerated at the Census of 1921 in each of the five social classes (column 2) must now be multiplied by their appropriate C.M.F., and these weighted populations (column 4) summed. It will be apparent, a priori, that in any district having a social distribution exactly similar to that of the country as a whole, the sum of the weighted populations must be equal to the actual numbers enumerated at the Census. If, however, the proportion of males classified to groups I and II be in excess of that pertaining to England and Wales (and the remaining social groups correspondingly in defect) the sum of the weighted population will be smaller than that actually enumerated. Conversely, a population relatively defective in males of the upper classes will have a weighted population in excess of that enumerated. To obtain the correcting factor for any disease group, then, the actually enumerated population must be divided by the sum of the socially-weighted population. The factor thus obtained, when in excess or defect of unity, denotes the existence in the district under consideration of a population constituted with respect to infant mortality (or any of its components) favourably or adversely respectively compared with the country as a whole (the 'standard' population). The crude rate of mortality in each district, stated per 1,000 births, is now multiplied by its correcting factor for social status, and we thus obtain a distribution of mortality rates for a series of districts for which variation of economic distribution have been stabilised, so far as the method permits this to be done. From the example furnished by Hampstead (table 34) we see, as is well known from other considerations, that this borough is well above the average social level, and that, reduced to the status of/

TABLE 35. Example of the effect of eliminating social differences.

	Crude rates			Corrected rates.		
	Hampstead	Poplar	Ratio $\frac{P}{H}$	Hampstead	Poplar	Ratio $\frac{P}{H}$
	Infant mortality	55.3	74.4	1.35	65.5	70.7
Infectious diseases	2.8	5.2	1.86	3.6	4.8	1.33
Tuberculosis	0.8	1.4	1.75	1.0	1.4	1.40
Diarrhoea	7.5	9.9	1.32	9.5	9.1	0.96
Congenital debility	23.3	29.3	1.26	26.0	28.4	1.09
Bronchitis and pneumonia	8.0	16.9	2.13	10.5	15.7	1.50

of the standard population, the rate of infant mortality would be 18.5 per cent. above that actually recorded, i.e. a rate of 65.5 compared with the crude rate of 55.3 per 1,000 births.

(2a) Reduction in variability of the rates of infant mortality:

For the purpose of testing how much closer to one another the rates of mortality would be in a given set of districts if the effect of varying economic status among them be eliminated, the ideal sample of districts chosen for investigation would be one in which as many as possible of the other factors influencing the rates of mortality in infancy could be deemed sensibly constant. From this point of view, the Metropolitan Boroughs of London form as near an approach to the desired sample as it is possible to obtain in this country. They are all essentially urban in character, the type of housing (as opposed to the amount of accommodation available) varies little, and geographical and climatic influences may be presumed constant for all. These therefore have been chosen for this investigation. Correcting factors have been calculated, as described above, for each borough for total infant mortality and for several groups of diseases; and comparison has been made between the conventional mortality rates and those found after correction for social status has been applied. Table 35 presents a contrast between the mortality experienced by the socially best and worst boroughs in the group, namely Hampstead and Poplar. In the triennium 1921-3, Poplar showed a rate from all causes under one year 35 per cent. in excess of that obtaining in Hampstead, but when allowance is made for the wide divergence in the social level of these two districts, this excess is reduced to 8 per cent. Similarly in the chief cause groups of death considered, much of the original difference between the two is effectively,
or/

or, in the case of diarrhoea and enteritis, completely eliminated. These figures suffice to show that in isolated instances we are able to effect a great reduction in the range of the mortality rates when suitable correction is applied for economic divergences.

But the real problem is a more generalised one. What in bulk is the difference in the amount of dispersion in the rates of mortality when correction is or is not made for social differences? Do we, in fact, after such correction tend to reduce all districts to a mortality experience sensibly equal? The only suitable statistical measure for furnishing an answer to such questions is the coefficient of variation, a constant which expresses the absolute variation in terms of the mean value of the rates of mortality. These, with their probable errors are given in table 36.

TABLE 36. Coefficients of variation
(Metropolitan boroughs).

	Crude rates.	Corrected rates.
Congenital debility etc.	11.09 ± 1.01	9.32 ± 0.84
Diarrhoea and Enteritis	27.05 ± 2.61	24.63 ± 2.34
Bronchitis and pneumonia	29.06 ± 2.83	25.55 ± 2.44
Infectious diseases	31.79 ± 3.14	28.95 ± 2.82
Tuberculosis (all forms)	39.62 ± 4.09	38.11 ± 3.90
Infant mortality	14.99 ± 1.38	12.58 ± 1.15

From these results it is clear that although in every group the variability is slightly reduced, in not one instance is it possible to attach statistical significance to the difference found. Thus in this sampled population, the tendency towards a levelling of the rates of mortality is a very slight one. Moreover, when we recollect that the various disease groups considered/

considered here are affected in different degrees by the conditions pertaining to differences in social status (as Table 33 readily shows), no evidence is contained in these figures that the differences in the dispersion of each of these rates would be eliminated if we considered only districts of the same level socially. Stated otherwise, the death rate from all forms of tuberculosis varies much more from place to place than, for example, that from congenital debility, and this greater variation is not at all because tuberculosis is more closely related than is congenital debility to social and economic factors.

(2b) Alteration in the relative position of the districts:

The 28 boroughs have been ranged in order from the lowest to the highest with regard to both the crude and corrected rates of infant mortality from all causes and from separate cause-groups. Correlations have then been deduced between the rank which each borough occupies in the distribution of crude and corrected rates. These are collected in table 37.

TABLE 37. Coefficients of correlation
(Metropolitan boroughs).

Infant mortality	0.951
Infectious diseases	0.970
Tuberculosis	0.992
Diarrhoea	0.945
Congenital debility	0.953
Bronchitis and pneumonia	0.978

It is at once clear from the magnitude of these coefficients that no great change has been effected in the position of the boroughs generally. Those which originally occupied favourable positions have still, after eliminating economic variations, the more desirable rank. The most noteworthy differences, which, it will be seen, are by no means very great, need only be summarised. The two figures after each borough mentioned relate/

relate to the position occupied when crude and corrected rates respectively are considered.

All causes. Finsbury (23, 18); Poplar (17, 10); Hampstead (2, 6); Holborn (20, 24); Greenwich (8, 4); St. Marylebone (12, 16).

Common infectious diseases: Finsbury (13, 9); Poplar (16, 11); Wandsworth (9, 13).

Tuberculosis: Hampstead (5, 8).

Diarrhoea and enteritis: Bermondsey (21, 15); Finsbury (18, 13); Poplar (11, 5); Hampstead (2, 9); Holborn (15, 21).

Congenital debility: Hampstead (2, 8); Poplar (23, 17).

Bronchitis and pneumonia: Poplar (21, 15).

Are we entitled to infer from these results that economic status has little intrinsic influence upon rates of mortality, or - to put it more bluntly - that if Mr. Bernard Shaw's ideal were realised and all family units enjoyed the same incomes, the variability of rates of infant mortality of the London boroughs would be much what they are now? I pointed out at the beginning that this method of standardisation rests upon an assumption which may lead us into error. Until I have had an opportunity of applying it more widely - it can obviously, now that we have the rich harvest of results contained in the new Decennial Supplement, be used to standardise mortality in all or any cause groups of death - I prefer to draw no more definite conclusion than that, in spite of the great contrasts between the rates of mortality of different economic groups, standardisation for economic status does not reduce variability so much as seemed a priori probable.

These findings, it should be pointed out, are in no way inconsistent with those already given (Section III). There it was shown that infant mortality (excluding the ante-natal death/

death rate) was significantly correlated with various measures of housing conditions and economic status; but if from these correlation results we deduce the statistical constant equivalent to what the process described in this section is measuring (namely, the reduction in variability of the rates of mortality which is effected by eliminating differences in housing and economic conditions) then the results are in great measure consistent one with another. The simple procedure outlined in this section, moreover, provides a rapid and (so far as any general measure of poverty available in official statistics is concerned) an accurate measure designed to free rates of mortality from the disturbing effects of differences in the economic position of the districts investigated. Its utility and importance in future statistical inquiries is thus comparable with that following the introduction into vital statistics of methods of correcting death rates for differences in age distribution of the population.

VIII - SUMMARY AND CONCLUSIONS.

In the present study the chief aim has been to inquire into the causes of the decline in the mortality of infants and to assess the relative importance of several influential factors at different periods of infant life (including the ante-natal). The chief findings may now be summarised.

In Part II it has been shown by an analysis of the changes in the course followed by the separate causes of infant death that the decline in mortality has practically been restricted to post-natal causes. The gastro-enteric infections, diarrhoea and abdominal tuberculosis, have shown the greatest relative decrease. Other causes in which a sensible amelioration has been shown are: overlying, measles, atrophy, debility and marasmus and infantile convulsions. Whooping cough, syphilis, bronchitis and pneumonia, premature birth and still-births have shown no tendency in the period under review to decline, whereas congenital malformations and injury at birth have definitely increased in frequency. No change of any importance could be demonstrated in the secular movement of the rate of maternal mortality (which in view of its relations to foetal and infant mortality has been subjected to analysis for the sake of completeness). The possible causes of the decline were then discussed, and the evidence submitted makes it probable that more adequate and more intelligent maternal care bestowed on the infant can best explain the changes in mortality found. Although it was admitted that administrative measures may have exerted an indirect (but certainly no direct) influence, there is much room for scepticism as to the part played by these in the improvement of the/

the infant death rate, and there is no evidence derivable from mortality statistics that the decline in infant mortality and the increase in welfare work etc. are causally-related phenomena. The most simple interpretation, and the one which we suggest as the most probable, is that the decline in the birth rate, probably due in great measure to deliberate restriction of the size of the family to a desired number, is in part of the expression of a praiseworthy attempt on the part of the parents to provide the family with the best prospect of survival and success in life.

Part III contains the results of a study of the mortality data in different areas of England and Wales in the most recent available period. Here it was shown that the still-birth rate is definitely related to indices of the availability of skilled medical assistance in confinement, but unrelated to external environmental conditions (housing and economic circumstances) and the health of the mother. The total effect of all of these three possibly influential factors will only account for a small fraction of the total number of still-births; and here the conclusion was advanced that the greater proportion of foetal deaths are not amenable to preventive measures in the present state of medical knowledge. (This is supported by the evidence of Part IV) Non-natal deaths also show definite association with variations in the provision of skilled medical assistance, indicating the probability that a substantial proportion of these deaths is within the scope of a more energetic obstetrical service. These deaths, moreover, contrary to general opinion, are definitely influenced by environmental conditions. The mortality at this period of life was found to be appreciably heavier in families in low economic circumstances and living under/

under conditions of overcrowding. The health of the mother could not be shown to be a factor of much importance to the infant at this early age. Here again, however, we cannot account for this neo-natal death rate wholly in terms of even all of these factors, and we submit that at least part of the remainder is at present uncontrollable. The post-natal death rate, even at its present low level, is the part of infant mortality to which we can still look for the greatest relative improvement. The health of the mother, we have been led to conclude, is of more importance than are differences in the less intimate surroundings of the infant. The great part of the mortality in post-natal life we have found to be explicable in terms of the more commonly recognised influential factors.

The causes of still-birth have been submitted to scrutiny in Part IV. The major causes in a random sample of the general population are developmental defects, unknown causes and prematurity. A point of importance, hitherto entirely overlooked, is that hospital statistics produce a far too optimistic outlook in this connection. The very nature of the hospital population will make it evident that those causes of still-birth in which the lives of both mother and foetus are endangered will be over-represented in frequency compared with the true frequency in a general population of childbearing women. Thus when we attempt to estimate the amelioration to the still-birth rate which is likely to be effected by more intense ante-natal and natal supervision and treatment of the mother, statistics from a hospital taken at their face value will lead us to expect a greater improvement than can possibly happen. The claims put forward as to the value of such methods, being too optimistic on this basis, will not be justified by future experience and may ultimately lead to an otherwise/

otherwise extremely efficacious measure being brought into disrepute. The majority of still-births are at present outwith human control:

Part V contains the results of a study of the statistics - anatomical features of foetuses and infants. The weights of five viscera in addition to body weight and length of a series of foetuses and of infants who had died in the first year of life have been subjected to analysis; and from this we have been able to show that the foetus is physically in a state of extreme instability, that the conditions which kill in utero are rapidly fatal, and that the male is more unstable than the female. During the first year of life this instability (as evidenced by visceral variation) tends to wear off gradually; so that some time is obviously necessary for the foetus to adapt itself to its entirely new mode of existence after birth. This physical state of unstable equilibrium in foetal and early infant life has been suggested as a further reason for the higher mortalities at these than at later years of childhood because of the greater ease with which adverse circumstances may upset a bodily mechanism, the parts of which are not yet fully coordinated.

Finally in Part VI is described a method for standardising rates of infant mortality for differences in the social and economic level of any series of districts. This provides us with an easily calculated and accurate arithmetic measure of how far any given difference from the average economic position will affect the rate of mortality; and by multiplying the crude death rates by the correcting factors found by the method suggested we reach rates from which the effects of the social and economic factor are eliminated.

Applied/

Applied to the statistics of the metropolitan boroughs we have shown actually how much smaller than is generally believed is the quantitative effect on rates of mortality of differences from the average economic position. The scope and advantages of the method have been set out in detail in the text.

In a sense, then, the conclusions to be derived from these researches are pessimistic. A great proportion of still-births and a somewhat smaller proportion of neo-natal deaths are not amenable to the measures generally advised. More thorough research into deaths especially from ill-defined and unknown causes but also into the causes and prevention of prematurity seems to be urgently called for. A certain proportion of deaths in early infant life are amenable to obstetric control, a smaller proportion should react to efficient ante-natal care; but little can be hoped for from improvements in the general surroundings (housing and economic) to which the mother is exposed during pregnancy, nor have we any reason to hope that the improvements now taking place in the general health of mothers will react favourably on the ante-natal death rate. Later in infant life, mortality can be further reduced by improvements in housing conditions, economic circumstances, parental health and, above all, maternal care. The administrative measures designed to effect such improvements are as yet in their infancy. With further extension of and cooperation between the various medical services, the outlook at this period of life in respect of mortality is comforting.

VIII APPENDIX NOTES.

1. Regression coefficient: A coefficient of regression may be regarded as a velocity - the rate of change per unit of time. In the case to which it is applied in this study (the decline in infant mortality), the coefficient represents a percentage decrease (or increase) in mortality from any cause per annum.

2. Coefficient of Correlation: For the non-statistical reader a coefficient of correlation may be regarded simply as an arithmetic measure of the degree of association between two variables. The values can range from 0 (in which case the two variables are said to be independent of one another) to ± 1 (in which case the variables are perfectly, i.e. causally related). Most of the values found are intermediate between 0 and 1; but the nearer to unity, the closer is the degree of association. A positive sign indicates that as one of the variables increases in value, the associated one also tends, on the average, to increase; a negative coefficient means that as one variable increases, the other tends, on the average, to decrease.

3. Coefficient of Partial Correlation: This is a measure used when more than two variables are considered and we desire to measure the closeness of association between any two of them apart from any indirect association which might be introduced by reason of these being both correlated with the remaining ones. For example, if we denote infant mortality by 1, maternal health by 2 and housing conditions by 3, we know that mortality is related to both health of mother and housing conditions and that maternal health is also/

also related to housing conditions. We may then wish to find what is the association between infant mortality and the health of the mother in a sample in which there were no differences of housing conditions. This can be found by determining the partial correlation, which in this case would be denoted by $r_{12.3}$.

4. Coefficient of Multiple Correlation: If we find that (e.g.) infant mortality is related to several individual factors, say as before, health of mother and housing conditions, we desire to know what is the quantitative degree of association between infant mortality and both of these other variables. Statistically this is measured by a coefficient of multiple correlation, and would be denoted in the above example by $R_{1.23}$.

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