PASTEURIZATION OF MILK AND INFANT MORTALITY RATES IN TORONTO, VANCOUVER, AND VICTORIA

BY

ALAN BROWN, M.D.

Professor of Paediatrics, University of Toronto

[In reply to an inquiry from Dr. Donald Paterson, Secretary of the British Paediatric Association, regarding statistics of infant mortality rates and pasteurization in Vancouver and Toronto, Dr. Alan Brown was able to collect through Dr. Bob Defries, Director of the School of Hygiene, University of Toronto, the following information, which he thinks presents the situation very plainly. It is, he says, quite unfair to compare statistics as statistics, because there are so very many factors to bear in mind. "We all know here that the climate is much more favourable on the Pacific coast than it is in the eastern part of the country. And, after all, the great importance of pasteurization lies in its reduction of all milk-borne infections. We have not had a case of milk-borne infection (including bovine tuberculosis) enter the Hospital for Sick Children—having lived in the city of Toronto and used nothing but pasteurized milk—since 1915." The inquiry arose out of a statement on infant mortality in Toronto made by Dr. A. H. Macdonald in a letter to the JOURNAL of Jan. 9, 1943.]

Discussion of pasteurization in the British Parliament was recorded in the *British Medical Journal* in September and December, 1942. In the issue of Dec. 19, 1942 (p. 727), there appeared a leading article entitled "Do Doctors Disagree about Pasteurization?" and several letters were subsequently published from correspondents. The following abstract from a letter from A. H. Macdonald, appearing in the correspondence columns of the *Journal* for Jan. 9, 1943 (p. 52), affords the probable basis of this inquiry from England regarding infant mortality rates and pasteurization in these Canadian cities: "The infant mortality in Toronto, where all milk is pasteurized, is double that in Victoria, Vancouver, where all milk is drunk raw. My own statistics dealing with large masses of children for 18 years show raw milk to be a good thing." (The letter refers to "Victoria, Vancouver." It is possible that the writer meant Victoria on Vancouver Island. As it is not clear, we have dealt with the cities of Victoria and Vancouver.)

Comment

1. The infant mortality rate for the city of Vancouver in 1940 was 25.6; for Victoria, 28.4; for Toronto, 38. The rate in Toronto is approximately one and a half times the Vancouver and Victoria rates.

2. The statement "in Victoria, Vancouver, where all milk is drunk raw," is incorrect, since in the report from the Medical Officer of Health for Vancouver, made to the Committee on Milk of the Canadian Public Health Association, as relating to 1938, the proportion of milk pasteurized in that city was given as 79%; and in a similar report from the Medical Officer of Health for Victoria 50% was stated to be pasteurized.

Discussion

In considering the causes, as recorded statistically, of infant mortality under the headings of (a) "Diseases Peculiar to Early Infancy"; (b) "Communicable Diseases"; (c) "Acute Respiratory Infections"; (d) "Diarrhoea and Enteritis"; and (e) "All Other Causes," for Toronto and Vancouver, reference will be made to the data for the years 1919, 1929, and 1939. These three 10-year periods indicate the significant changes.

(a) *Diseases Peculiar to Early Infancy*.—In 1919 Toronto had a rate of 41.2 for this group of causes, in contrast to Vancouver, which had a rate of 26.3. Ten years later (1929) the Toronto rate had fallen to 37 and the Vancouver rate to 16.3. In 1939 the Toronto rate had fallen to 25.7 and the Vancouver rate to 13.1. The Toronto rate is practically double the Vancouver rate. As this group of causes represents more than half of the total infant mortality it is evident that much of the difference between the rates for Toronto and Vancouver is due to this group of causes, which includes prematurity, etc.

(b) *Communicable Diseases*.—The rate in 1919 for this group of causes was 7.4 in Toronto and 5.2 in Vancouver. Ten years later (1929) the Toronto rate fell to 5.7 and the Vancouver rate to 1.6. In 1939 the Toronto rate was 1.5 and the Vancouver rate 0.5. It will be noted that the rate in Toronto is three times the rate in Vancouver; but the total number of deaths from communicable diseases is quite small and does not explain the difference between the gross infant mortality rates.

(c) *Acute Respiratory Infections*.—In Toronto the rate in 1919 for this group of causes was 16.3, and in Vancouver 7.4. In 1929 the Toronto rate was 6.5 and the Vancouver rate 5.7. In 1939 the Toronto rate was 4.8 and the Vancouver rate 2.1. It will be noted that the Toronto rates are again almost double the Vancouver rates; but the total number of deaths contributed by this group of causes is small and does not represent a major factor in the difference between the two rates.

(d) *Diarrhoea and Enteritis*.—In 1919 the Toronto rate was 11.5 and the Vancouver rate 5.2. Ten years later (1929) the rate for Toronto had fallen to 9.7 and for Vancouver to 1.4. In 1939 the rate in Toronto was 2.2 and in Vancouver 0.

(e) *All Other Causes*.—In 1919 the rate in Toronto was 25 and in Vancouver 12.6. In 1929 the rate in Toronto was 19.3 and in Vancouver 8.5. In 1939 the rate in Toronto was 9, and the rate in Vancouver also was 9.

From this analysis it is evident that the major differences in the rates in Toronto and Vancouver are accounted for largely by the differences in the rate in the group of causes entitled "Diseases Peculiar to Early Infancy." It is interesting to note that the fall in death rate in the group of diseases to which pasteurization would primarily be related—namely, diarrhoea and enteritis—is now approximately the same in both cities and represents a very small number of deaths.

Reference is not made to the city of Victoria because data of infant mortality rates by groups of causes were not available and, further, because the figures are for Greater Victoria, which includes adjacent municipalities. As, however, the infant mortality rate for Victoria is only slightly higher than that of Vancouver, analysis would probably yield similar data.

Graph showing infant mortality rates in Toronto, Victoria, and Vancouver from 1914 to 1940.

In considering this question of the difference in infant mortality rates of certain cities on the Pacific coast it is of interest to refer to a paper by Bellows and Reed published in 1934 in the *American Journal of Hygiene* (vol. 20, p. 565) entitled, "The Effect of Certain Environmental Factors on Urban Infant Mortality Rates." In this article a table is presented giving the crude infant mortality rates for cities in the United States having a population of over 100,000 and under 1,000,000, corrected for five factors—temperature, latitude, relative number of foreign-born, relative number of coloured, and unemployment in manufacturing and mining. Of the seven cities with the lowest crude infant mortality rate, six were on the Pacific coast, Seattle leading with the rate of 56.6. To each of these a substantial correction was considered necessary, ranging from plus 9 to plus 28. Seattle and Portland, Ore., retained the lead among the first seven; in fact, among the first ten. Others on the coast did not fare so well. "The first ten cities whose crude rates were all below 80 have marked increases in their corrected rates. The group will be seen to include all of the cities located on the northern Pacific coast, where climate is relatively favourable. Allowance for this factor, and for the lack of industrialization in the Pacific cities, was largely responsible for the changes in their rates. Adjustment for the small industrial development was the principal

correction in the other cities in this low mortality group (the first ten), temperature in this case being a factor of minor importance."

This subject was further considered by Dr. L. A. Pequegnat, Deputy Medical Officer of Health, Toronto, who presented a paper entitled "The Present Problem in Infant Mortality" to the Academy of Medicine, Toronto. The paper was published in the *Canadian Public Health Journal* (1938, 29, 477). It reproduces a table of Bellows and Reed's. In this paper the rates for Toronto and Vancouver are presented for the years 1930 to 1935. In 1930 and 1935 the infant mortality rate in Toronto was double the Vancouver rate. It would seem, therefore, that the lower rates of Vancouver and Victoria require in their consideration some of the factors studied by Bellows and Reed. That climate alone is not the factor is evidenced by the fact that the rate in Calgary in 1940 was 31.2 and in Edmonton 30.6, in comparison with 25.6 in Vancouver and 28.4 in Victoria. Figures for other cities in Canada are: St. John, N.B. (65% of milk pasteurized), infant mortality rate 55 (1940); Montreal (95% pasteurized), rate 70 (1939) and 58 (1940); Three Rivers, Que. (60% pasteurized), rate 97 (1940). In Ontario all municipal milk supplies were required to be pasteurized in 1938. Data for 1940 give the following infant mortality rates: Hamilton 33, London 36, Kitchener 37, Ottawa 49.

From these rates it is evident that neither climate nor pasteurization of milk gives an answer to the differences in rates. The larger number of deaths due to prematurity, etc., is an important factor.

THE OCULAR CRITERIA OF DEFICIENCY OF RIBOFLAVIN

BY

M. K. GREGORY, M.B., Ch.B., D.O. Oxon

Since Bessey and Wolbach (1939) showed that corneal vascularization was a sign of riboflavin deficiency in rats, and since the publication of two papers on ocular manifestations of ariboflavinosis, by Kruse *et al.* (1940) and Sydenstricker *et al.* (1940), there has been a spate of enthusiastic but uncritical investigations. It is therefore necessary that there should be some attempt to clarify the confusion of thought and to correct the looseness of terminology that has arisen. Evidence of this confusion is to be found, for example, in a recent report of an investigation by Scarborough (1942) in which he has taken the sign of circumcorneal injection as being of primary importance. This sign can occur in early ariboflavinosis, but it certainly is not pathognomonic; and I do not find anything in the paper by Sydenstricker *et al.* (1940), which is quoted by Scarborough, to indicate that this isolated sign should be so interpreted. A further discrepancy which I have noted occurs in the two papers mentioned above, which in one place describe the earliest change noted as being a superficial invasion of the cornea by capillaries, and in another place say that "the earliest and most common sign of ariboflavinosis is circumcorneal injection." Also the terms "circumcorneal injection" and "ciliary congestion" are used indiscriminately without indicating whether just some degree of fullness of the superficial pericorneal vessels is meant or whether there is an intense ciliary blush. From my own recent observations and from verbal corroboration by other observers I question whether what is clinically known to ophthalmologists as circumcorneal injection is necessarily an accompaniment of mild early corneal vascularization as we see it in this country.

As the ocular changes in early deficiency of riboflavin are concerned with the vascular supply of the limbus, it is essential that observers first should have an exact knowledge of the normal blood supply and arrangement of vessels at the limbus;

secondly, they should have an accurate idea of the changes which, in the light of our present knowledge, may be expected to occur; and, thirdly, they should know what other conditions may produce somewhat similar appearances and how these may be distinguished. I propose to give a brief outline of these three points.

Fortunately it is rare at present for nutritional deficiencies to be more than slight in this country, and therefore, as it is the mild rather than the gross changes that are being considered, the picture that we may expect to find will scarcely be comparable with the fully developed cases described by Sydenstricker *et al.* (1940) and Kruse *et al.* (1940). I am not considering such signs as pigment deposits on the iris and mydriasis, as the significance of these has not yet been adequately established.

The Normal Blood Supply of the Limbus

The blood supply of the pericorneal region comes from the anterior ciliary arteries arising indirectly from the ophthalmic arteries via the arteries of the four recti muscles, each tendon being accompanied by two, except the external rectus, which has only one. These arteries pass forward on the episclera, giving branches to the sclera, corneal margin, and conjunctiva. The main branch of each artery pierces the sclera about 4 mm. from the corneal margin to enter the ciliary muscles, where it anastomoses with the long posterior ciliaries to form the *circulus iridis major*. Before piercing the sclera the anterior ciliary arteries give off the anterior conjunctival branches, which run forward deep to the posterior conjunctival arteries and anastomose to form the pericorneal plexus, consisting of a series of arcades parallel with the corneal margin. These give off final branches, marginal loops which bend round on their tracks to form venules leading into a venous plexus built up on similar lines. The anterior conjunctival arteries also give off small posterior branches to anastomose with the posterior conjunctival arteries, which come from the palpebral branches of the nasal and lacrimal arteries of the lids.

The pericorneal plexus is in two layers—the superficial conjunctival and the deep episcleral. This distinction is important because in superficial affections of the cornea the superficial portion is injected and may rightly be termed circumcorneal injection, whereas in diseases of the iris, ciliary body, or deep part of the cornea the deep episcleral portion becomes congested and gives rise to the rose-pink or violet band typical of ciliary congestion. This deep ciliary congestion is always a serious sign as indicative of uveal disease, but superficial circumcorneal injection can be transiently produced by vigorously rubbing the eye for a moment; and it results from irritations such as exposure to wind, cold, bright light, mild chemical irritants, mild infection, and numerous other causes. From such causes there must be a considerable proportion of the population showing circumcorneal injection, which would fit the description given in the paper by Sydenstricker *et al.* (1940) as "often it was grossly visible, frequently it could be seen with a hand lens or ophthalmoscope, and always it was obvious on slit-lamp inspection as marked congestion and proliferation of the limbic plexus." On this point I would ask what evidence there is that there is any real proliferation and not only engorgement of pre-existing but empty and therefore invisible vessels.

The extent to which the vessels may, within normal limits, extend on to the cornea should also be defined. The limbus is the whole zone in which the sclera and cornea overlap owing to the edge of the cornea being inset like a watchglass in a groove in the sclera. Though it is said to be 1 mm. in width, it is very variable, often being much wider than this. With the slit-lamp the whole width of the limbus appears like ground glass, increasing in opacity peripherally. It may be segmented by digitations or may appear homogeneous. The marginal plexus is described as occupying a triangular area whose apex lies where Bowman's membrane ends and whose base is formed by episcleral and scleral tissue (Wolff, 1940). Thus it is normal for vessels to occupy the whole width of the limbus, and there is not necessarily an avascular zone between the plexus and sclero-corneal junction; and capillaries which do not extend beyond the limbus thus defined are considered to be within the limits of normal.